VOLUME 51 NUMBER 6

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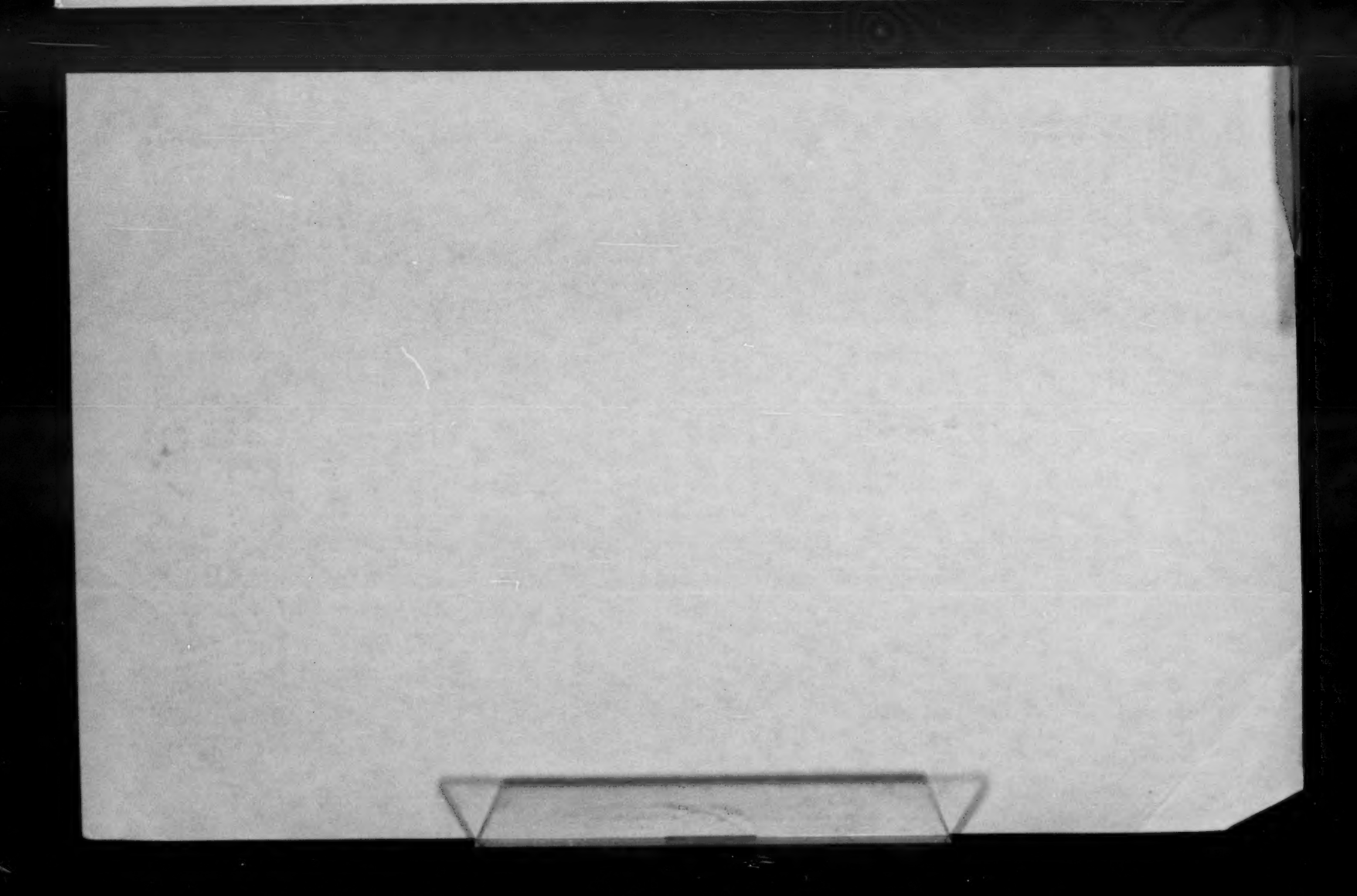

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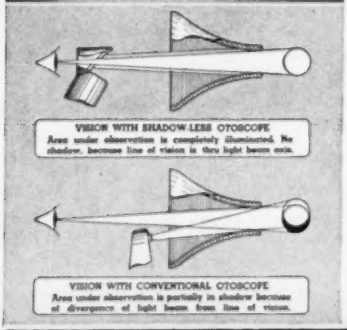
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## Effects of Hearing Defects on Personality

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### NO BIRD SINGS FOR THE HARD-OF-HEARING

In the world of the deaf "many things are in motion, but there is no sound." [1] The brook rushes over stones, but does not gurgle, the leaves move on the trees but do not rustle, the robin outside of the window has its mouth open, its throat pulsing, but its goodnight song is not audible. The deaf hear none of these soul-lifting sounds which those with normal hearing take for granted.

The hard-of-hearing, like the totally deaf, are deprived of the pleasant stimulation of nature's voices. As hearing loss progresses the voices become fainter and fainter, until at last none at all is heard.

This loss cannot be without its effect upon personality. There is a frustration in seeing sound rather than hearing it, a frustration which undoubtedly contributes to the "despondence, feeling of incalculable loss, hopelessness" which are among the personality changes noted in the hard-of-hearing.[2]

This feeling of frustration can, of course, be overcome with proper psychological help. Nor need any except the totally deaf live in a soundless world, since mechanical means are available to amplify residual hearing. The hard-of-hearing can, and should, be given proper guidance so they can enjoy, at least in part, the stimulating world of natural sounds.

### THE PERSONALITY OF THE HARD-OF-HEARING CHILD SELDOM FLOWERS FREELY

A child's personality can develop freely only in an atmosphere of the calm love of his parents, in full social contact with his contemporaries, in satisfactory achievements at school. Often too young to realize there is an organic cause for the lack of intelligibility of the words he hears, unable to explain to others the nature of his difficulties, he finds himself criticized, accused of being inattentive, publicly reprimanded or even physically punished. His companions may scorn or deride him.[3]

Physical fatigue comes from the strain of constant efforts at concentration,[3] a fatigue which causes the intelligent child to retreat into fantasy, the retarded child to fidget or rebel.[4]

When the physician is consulted because the child becomes easily tired—or indeed for any other reason—it is important that he keep in mind the possibility that a hearing defect may be present.

Even when parents recognize the handicap under which their child struggles, they may, by their very excess of care, contribute to his personality problems. They may pity him, wait on him, anticipate his needs, keep him in a state of infantility.[5]

What is needed is the re-education of parents (often suffering from a greater emotional imbalance than their child)[6] so that they will give him their understanding and encouragement. They must induce the hard-of-hearing child "to speak and to listen" [6] for upon this depends, "his future adjustment to the larger world, his emotional stability, and his urge to understand other people." [6] And, of course, the mechanical means which science has devised to aid his hearing must be made available to him.

### THE HARD-OF-HEARING ADULT HAS HIS PERSONALITY PROBLEM, TOO

On the adult's adjustment to his loss of hearing depends the happiness which he may obtain from life. When hearing loss is sudden, as from concussion or physical trauma, there may be the feeling that life is forever blighted. Such acute reactions may respond more readily than those which are due to the long-standing hearing defect.[3]

First step in adjustment is the recognition that the hearing loss is a permanent one (when this has been definitely determined); both the patient and his advisers must face this fact before proper emotional adjustment can take place.[7]

With acceptance of his disability, with proper assistance in learning to overcome his handicap, the hard-of-hearing patient will be brought back from his silent world into the world of his friends. Always it should be remembered that "there is rarely a deaf or hard of hearing person for whom something cannot be done educationally." [6]


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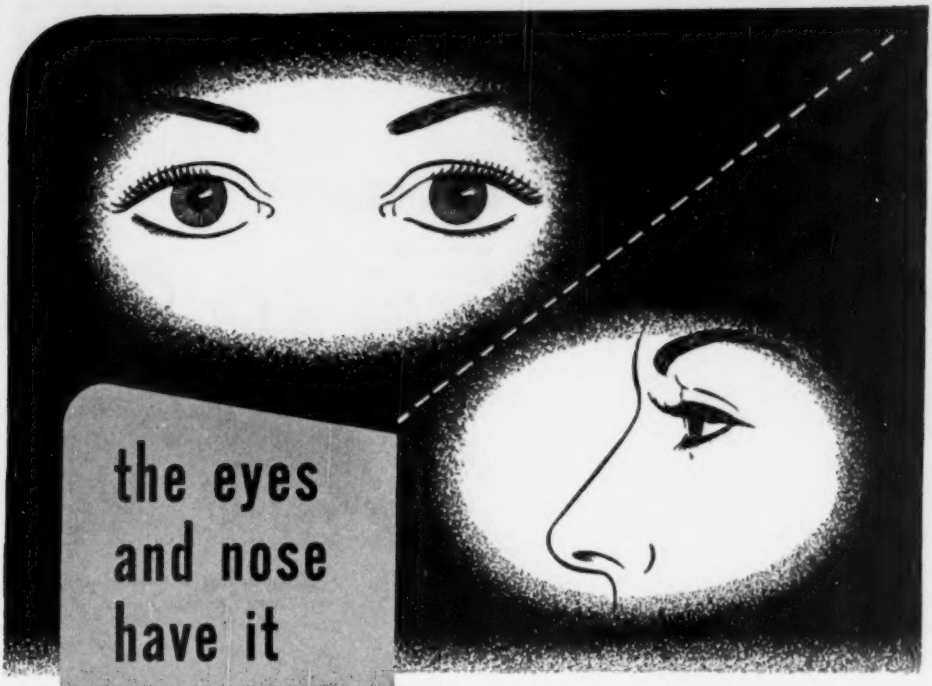


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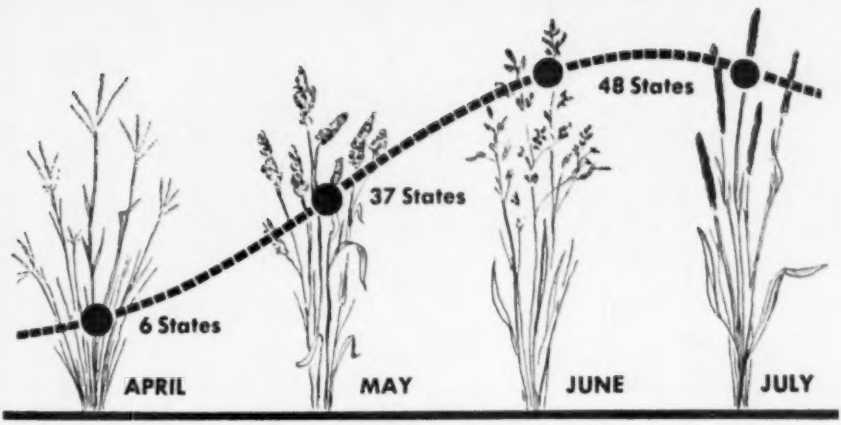


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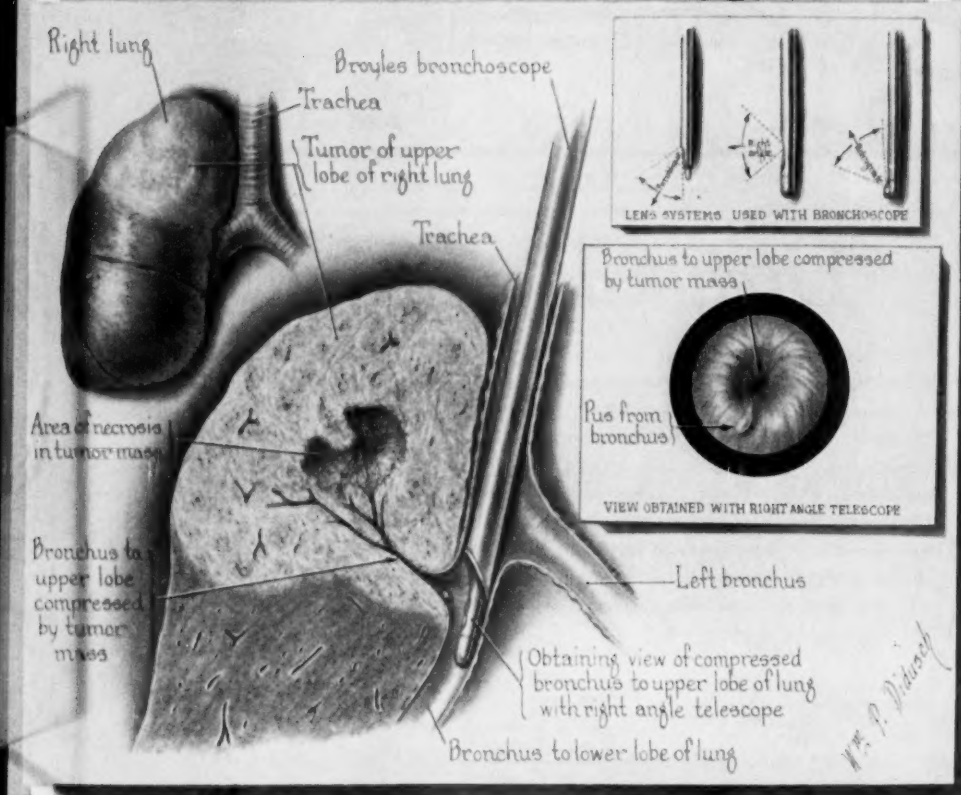
Right lung
Broyles bronchoscope
Trachea
Tumor of upper lobe of right lung
Trachea
Area of necrosis in tumor mass
Bronchus to upper lobe compressed by tumor mass
Left bronchus
Obtaining view of compressed bronchus to upper lobe of lung with right angle telescope
Bronchus to lower lobe of lung
LENS SYSTEMS USED WITH BRONCHOSCOPE
Bronchus to upper lobe compressed by tumor mass
Pus from bronchus
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1950

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# CONTENTS OF VOLUME 51

## JANUARY 1950. NUMBER 1


PAGE

Plight of the Nerve-Deaf Patient: The Uselessness of All Present Therapy; the Practical Usefulness of Aural Rehabilitation. L. E. Morrissett, M.D., Greenwich, Conn. ..... 1

Peripheral Facial Paralysis in Fractures of the Temporal Bone: Indications for Surgical Repair of the Nerve; Report of Cases in Which the Ballance and Duel Operation Was Used. Karsten Kettel, M.D., Hillerød, Denmark 25

Prevention of Deafness. Edmund Prince Fowler, M.D., New York..... 42

Superficial Epidermoid Carcinoma of the Larynx. Hans Brunner, M.D., Newark, N. J. ..... 49

Palatal Myoclonus: Report of Three Cases. Victor R. Alfaro, M.D., Washington, D. C. ..... 65

Herpes Zoster Oticus (Ramsay Hunt's Syndrome): Comments on an Article by Johnson and Zonderman. Kurt Tschiassny, M.D., Cincinnati..... 73

Dacryocystorhinostomy: Report of One Hundred and Fifty Cases of Dacryocystitis Occurring from 1938 to 1947. Bernard Welt, M.D., Brooklyn.... 83

New Neurosurgical Approach for Treatment of Otosclerosis. Manuel Bordes-Valls, M.D., Valencia, Spain..... 96

Case Reports:

Cardiospasm with Thoracic Stomach and Congenital Shortening of the Esophagus. Eric Gutteridge, M.D., F.R.C.S. (Edin.), F.R.A.C.S., Melbourne, Australia ..... 102

Stenosis of the External Auditory Canal. Arthur L. Juers, M.D., and Charles F. Whitaker, M.D., Chicago..... 104

Progress in Otolaryngology. Summaries of the Bibliographic Material Available in the Field of Otolaryngology:

Otitis Media and Complications. B. R. Dysart, M.D., Pasadena, Calif. 106

News and Comment..... 124

Abstracts from Current Literature..... 125

Society Transactions:

Chicago Laryngological and Otological Society.....133

College of Physicians of Philadelphia, Section on Otolaryngology..... 143

Directory of Otolaryngologic Societies..... 148


## FEBRUARY 1950. NUMBER 2


Ménière's Syndrome: Observations on Vitamin Deficiency as the Causative Factor; III. The General Disturbance. Miles Atkinson, M.D., F.R.C.S. (Eng.), New York..... 149

Stricture of the Esophagus Associated with Operation for Duodenal Ulcer. Gerhard D. Straus, M.D., Milwaukee..... 165

Mucocele of the Frontal and Ethmoid Sinuses: Evaluation of the Use of Acrylics and Tantalum in the Surgical Treatment. Samuel Kaplan, M.D.; Ariah Schwartz, M.D., and Bates F. Metson, M.D., Van Nuys, Calif..... 172

Thrombosis of the Cavernous Sinus: A New Evaluation and Report of a Case with Recovery. Louis K. Elfman, M.D., Philadelphia..... 188

Chronic Indurative (Cervical Myalgic) Headache. Ralph W. Stevens, M.D., Walla Walla, Wash..... 196

Mechanics of the Nose, with Special Reference to Nasopharyngeal Discharge. G. Edward Tremble, M.D., Montreal, Canada..... 205

Influence of Electric Punch Card Machines on the Human Ear. Hugo Ribeiro de Almeida, M.D., São Paulo, Brazil..... 215

The Oncocyte in Nasal Mucous Membrane: A Study of an Unusual Epithelial Cell. Joseph G. Schoolman, M.D., Chicago..... 223

Cranial Chordoma. Joseph Freeman, M.D., New York..... 237

FEBRUARY—*Continued*

PAGE

Fistula Auris Congenita. Jack B. Miller, M.D., Portland, Ore., and Paul M. Moore Jr., M.D., Cleveland ..... 245

Case Reports:
Plasmocytoma of the Nasal Cavity. Thomas A. Maguda, M.D., and Sydner D. Maiden, M.D., Memphis, Tenn. ..... 258
Plasmocytoma: An Unusual Case with Progressive Involvement of Waldyer's Ring and the Larynx, Bronchus and Pleura. Walter L. Mattick, M.D., Buffalo ..... 263
Roentgenograms of Mediastinal Abscess. Eric Gutteridge, M.D., Melbourne, Australia ..... 272

Abstracts from Current Literature ..... 273

Society Transactions:
International Audiology Conference ..... 288
Chicago Laryngological and Otological Society ..... 294

Directory of Otolaryngologic Societies ..... 306

MARCH 1950. NUMBER 3

The Cochleogram and Its Clinical Application: Concluding Observations. Julius Lempert, M.D., New York; Philip E. Meltzer, M.D., Boston; Ernest Glen Wever, Ph.D., and Merle Lawrence, Ph.D., Princeton, N. J. ... 307

Ménière's Syndrome: A New Drug for Control of the Acute Attack. Miles Atkinson, M.D., F.R.C.S. (Eng.), New York ..... 312

Osteoma of the Frontal Sinus: Report of Five Cases. Lieutenant Colonel Norman E. King, Medical Corps, United States Army ..... 316

Surgical Decompression of Malignant Exophthalmos. Oscar Hirsch, M.D., Boston ..... 325

Role of Streptomycin in Laryngeal Tuberculosis. George E. Lieberman, M.D., and William A. Lell, M.D., Philadelphia ..... 335

Noise-Induced Hearing Loss. D. E. Wheeler, Ph.D., Iowa City ..... 344

Transtympanic Technic in Surgical Otology: Transtympanic Postscripts. Otto Popper, M.B., Ch.B., F.R.C.S. (Edin.), Johannesburg, Union of South Africa ..... 356

Melkersson's Syndrome. Francis H. McGovern, M.D., Danville, Va. ..... 371

Pressure Changes and Barotrauma Resulting from Decompression and Recompression in the Middle Ear of Monkeys. Hsiang-Tung Chang, Ph.D.; Rodolfo Margaria, M.D., and Samuel Gelfan, Ph.D., New Haven, Conn. 378

Epidermoid Tumors of the Frontal Bone, Sinus and Orbit. E. A. Thacker, M.D., Everett, Wash. ..... 400

Sarcoidosis (Boeck's Sarcoid) of the Upper Respiratory Tract: Report of a Case with Ten Years' Clinical Observation. David L. Poe, M.D., and Paul S. Seager, M.D., New York ..... 414

Case Reports:
Acute Otitis Media Due to Klebsiella Pneumoniae Treated with Chloramphenicol (Chloromycetin®). Elmer A. Friedman, M.D., Chicago ... 420

Progress in Otolaryngology. Summaries of the Bibliographic Material Available in the Field of Otolaryngology:
Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Inner Ear: Review of 1947 Literature. Arthur L. Juers, M.D.; Eugene L. Derlacki, M.D., and George E. Shambaugh, M.D., Chicago ..... 422

News and Comment ..... 438

Abstracts from Current Literature ..... 439

Society Transactions:
American Laryngological Association ..... 451

Book Reviews ..... 461

Directory of Otolaryngological Societies ..... 464

## APRIL 1950. NUMBER 4

PAGE

Collapsed Ala: Pathologic Physiology and Management. Samuel Fomon, M.D., and Joseph G. Gilbert, M.D., New York; Armand L. Caron, M.D., Worcester, Mass., and Samuel Segal Jr., M.D., Springfield, Mass. ..... 465

Bone Conduction in Otosclerosis. R. R. Woods, M.D., Dublin, Eire ..... 485

Some Effects of the Common Cold on Speech. Charles Lightfoot, Ph.D., Los Angeles ..... 500

Antireticular Cytotoxic Serum in Pediatric Otolaryngology. Edgar I. Steinberg, M.D., Philadelphia ..... 514

Sialography. F. Johnson Putney, M.D., and Myron J. Shapiro, M.D., Philadelphia ..... 526

Acute Subdural Empyema Secondary to Frontal Sinusitis: Report of a Case with Recovery. Joseph A. Mufson, M.D., and Marvin Wagner, M.D., Milwaukee ..... 535

Base of the Nose: Anatomy and Plastic Repair. Bruno L. Griesman, M.D., New York ..... 541

Endolymphatic Hydrops Without Vertigo: Its Differential Diagnosis and Treatment. Henry L. Williams, M.D.; Bayard T. Horton, M.D., and Lois A. Day, M.D., Rochester, Minn. ..... 557

Allergy of the Ear, with Classification and a New Treatment of Labyrinthine Hydrops by Dehydration. Arthur Dintenfass, M.D., Atlantic City, N. J. ..... 582

Endaural Radical Mastoidectomy for Chronic Mastoiditis. R. J. McQuiston, M.D., Indianapolis ..... 596

Case Reports:

Extramedullary Plasma Cell Tumor. Frederick Fuerste Jr., M.D., Milwaukee, and Sam S. Zuckerman, M.D., Cheyenne, Wyo. ..... 608

Fracture of Stomach Magnet: An Unusual and Interesting Case of Fracture of Magnet in Attempted Removal of Foreign Body from Stomach. Morris S. Bender, M.D., and Sam Clayton, M.D., D.D.S., Jamaica, N. Y. ..... 616

News and Comment ..... 619

Abstracts from Current Literature ..... 621

Society Transactions:

Chicago Laryngological and Otological Society ..... 629

Book Reviews ..... 638

Directory of Otolaryngologic Societies ..... 640

## MAY 1950. NUMBER 5

Bone Conduction Through the Auditory Ossicle Chain. Philip G. MacDonald, M.D., New York ..... 641

Fold and Crypt Formation in the Nasopharynx: An Anatomic and Clinical Study. Hans Lion, M.D., Cincinnati ..... 655

Threshold of Feeling in the Fenestrated Ear. Maurice Saltzman, M.D., and Matthew S. Ersner, M.D., Philadelphia ..... 667

Forced Circling Movements (Adversive Syndrome): Correction with Dimenhydrinate ("Dramamine"). M. Schiff, M.D., M.Sc. (Med.); W. G. Esmond, B.S., and H. E. Himwich, M.D., Ph.D., Medical Division, Army Chemical Center, Md. ..... 672

Heating of the Human Maxillary Sinus by Microwaves. John J. Ballenger, M.D., and Stafford L. Osborne, Ph.D., Chicago ..... 678

Speech Rehabilitation of Patients with Cleft Palate. Esti D. Freud, New York 685

Cancer of the Larynx Classified in Three Dimensions: An Aid in Management. Samuel Kaplan, M.D., Van Nuys, Calif. ..... 696

Tracheobronchial Aspiration Following Tonsillectomy with General Anesthesia. Clarence H. Steele, M.D., Kansas City, Kan., and Jack R. Anderson, M.D., New Orleans ..... 699

MAY—*Continued*

PAGE

Selection of Patients for Fenestration Surgery. C. M. Kos, M.D., and S. N. Reger, Ph.D., Iowa City ..... 707
Carcinoma of the Ear. Charles E. Towson, M.D., and William H. Shofstall, M.D., Philadelphia ..... 724
Site of Lesion in Paralysis of Twelfth and/or Seventh Cranial Nerve. Kurt Tschiassny, M.D., Cincinnati ..... 739
Origin and Treatment of Osteomas of the Paranasal Sinuses. Olav Erik Hallberg, M.D., and Joseph W. Begley Jr., M.D., Rochester, Minn. ..... 750
Clinical Notes; New Instruments and Technics:
A Method of Preserving Foreign Body Specimens Recovered by Endoscopy. Ernest B. Emerson Jr., M.D., Rochester, N. Y. ..... 761
..... 763
News and Comment ..... 764
Abstracts from Current Literature ..... 773
Society Transactions:
Chicago Laryngological and Otological Society ..... 779
Book Reviews ..... 780
Directory of Otolaryngologic Societies ..... 781

JUNE 1950. NUMBER 6

Histamine in Treatment of Certain Types of Headache and Vertigo Following the Fenestration Operation. George E. Shambaugh Jr., M.D., Chicago ..... 786
Use of Speech Tests for Evaluation of Clinical Procedures. S. Richard Silverman, Ph.D., St. Louis ..... 798
Clinical Application of Bone Conduction Audiometry. Raymond Carhart, Ph.D., Evanston, Ill. ..... 809
Masking and Shadow Hearing in Bone Conduction. Maurice Saltzman, M.D., and Matthew S. Ersner, M.D., Philadelphia ..... 814
Hoarseness in Children. John M. Loré Jr., M.D., New York ..... 826
Streptomycin in Treatment of Tuberculous Otitis Media: An Evaluation. Leon L. Titche, M.D., Tucson, Ariz. ..... 831
Diagnosis of Intracranial Lesions by Vestibular Tests. Fernando G. Santamarina, M.D., Habana, Cuba ..... 852
Histologic Picture of Inherited Nerve Deafness in Man and Animals. Franz Altmann, M.D., New York ..... 891
Transantral Approach for Excision of Sphenopalatine Ganglion for Intractable Facial Pain. David L. Poe, M.D., New York ..... 901
Otosclerosis: Résumé of the Literature with Clinical Observations. V. S. Subramanian, M.B., M.Sc. (Med.), Madras, India ..... 917
Thrombosis of the Cavernous Sinus. Will P. Pirkey, M.D., Baton Rouge, La. 925
Case Reports:
Tumor of the Glomus Jugularis. D. A. De Lisa, M.D., Schenectady, N. Y. ..... 928
Clinical Notes; New Instruments and Technics:
Ear Aspirator and Injector. Barnard C. Trowbridge, M.D., D.Sc. (Med.) Kansas City, Mo. ..... 930
..... 935
Abstracts from Current Literature ..... 936
News and Comment ..... 946
Society Transactions:
Chicago Laryngological and Otological Society ..... 948
Book Reviews ..... 
Directory of Otolaryngologic Societies ..... 

ARCHIVES OF OTOLARYNGOLOGY

VOLUME 51 JUNE 1950 NUMBER 6

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# HISTAMINE IN TREATMENT OF CERTAIN TYPES OF HEADACHE AND VERTIGO FOLLOWING THE FENESTRATION OPERATION

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CHICAGO

HISTAMINE for the treatment of vertigo was first reported by Alfoldy in 1938 and for the treatment of headache by Horton, MacLean and Craig in 1939.

Horton described a distinct clinical syndrome that he named histaminic cephalalgia, or vascular headache, that could be elicited by a subcutaneous dose of histamine and relieved by repeated injections of graduated doses of histamine given subcutaneously twice daily.

Hansel also used histamine in the treatment of cases of typical histaminic cephalalgia, but in contrast to the relatively large frequent subcutaneous injections advised by Horton, Hansel used an extremely minute dose of histamine given less frequently, once or twice a week, and usually supplemented by drops of very dilute histamine solution under the tongue twice daily.

My associates and I have followed the plan of treatment suggested by Hansel, and in typical cases of histaminic cephalalgia the results have been satisfactory in the majority of cases.

I have also used similar minute doses of histamine with certain patients in whom a group of symptoms developed in the ear operated on months or sometimes years after a fenestration operation. These symptoms consist of the rather abrupt onset, for no apparent reason, of a roaring or pulsating tinnitus in the ear operated on, with an uncomfortable sense of fulness or pressure; definite though usually mild vertigo on sudden movements; a distinct drop in hearing in the ear operated on, and headache or pain in or around that ear. One or several of these symptoms may be present at the same time in any one case, and occasionally all five will be noted at once. In some cases, after a few days or weeks, there may be a spontaneous remission, but with a tendency to recurrence. In other cases the symptoms persist for weeks or months. Examination of the ear discloses no local pathologic change to account for the symptoms. The administration of dilute histamine

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solution according to the method of Hansel frequently results in improvement of or complete relief from symptoms which in some cases have persisted for weeks or months without a spontaneous remission.

I have administered histamine in minute doses in 703 cases of fenestration, comprising about one third of all my fenestration cases. In 333 cases the results of therapy were inconclusive, owing to incomplete records; in 370 the results were definite, the symptoms being improved or relieved immediately or within a few days in 225, or 61 per cent, and not improved in 39 per cent. The effective dose of histamine was oftenest 0.1 cc. of 1 : 100,000,000 dilution by subcutaneous injection, but in a few cases this dose aggravated the symptoms and the effective dose was 0.1 cc. of 1,000,000,000; while in others it was 0.1 cc. of a 1 : 10,000,000 or 1 : 1,000,000 dilution. As a rule, if histamine is going to help, the relief from symptoms is prompt and definite after the first injection Occasionally a single injection suffices; oftener several are

TABLE 1.—*Frequency of Symptoms in 370 Patients That Received Histamine with Definite Results*

| Symptoms | Number of Patients | Per Cent with Relief or Improvement |
|---|---|---|
| Dizziness | 135 | 65 |
| Tinnitus | 120 | 60 |
| Drop in hearing | 72 | 35 |
| Pain | 43 | 90 |

required to secure lasting results. In some cases the histamine drops under the tongue seemed necessary to prevent a recurrence of symptoms.

Particularly interesting is the observation that some patients who got good results with histamine at first failed to receive benefit from the same therapy for the same symptoms recurring months or years later—in other words, histamine therapy sometimes tends to lose its effect.

The frequency of symptoms and the percentage of patients that were relieved by therapy with dilute histamine solution is shown in table 1.

A few typical case reports will illustrate this postoperative syndrome that may occur months or years after a fenestration operation.

### REPORT OF CASES

CASE 1.—J. F., a woman aged 48, had a fenestration operation in July 1943 that brought her hearing to the practical level, where it remained for three and one-half years. In December 1947 a "terrible" roaring developed in the ear operated on, accompanied with slight dizziness on stooping; a sense of fulness "as though there was a block of cement" in the ear operated on, and a drop in hearing. No local pathologic change was found in the ear to account for the symptoms. In February, these symptoms having persisted for two months, she was given a

single injection of 0.1 cc. of a 1:100,000,000 dilution of histamine phosphate subcutaneously, with 2 drops of 1:10,000,000 dilution under the tongue twice daily. Eleven days later the roaring was nearly gone and the "block of cement" was gone from the ear, but the dizziness and hearing were no better. The patient was told to take the same dose of histamine once a week for four weeks, with the drops under the tongue twice daily. One month later all the symptoms were relieved and her hearing was back at the practical level.

CASE 2.—D. F., a man aged 33, had a fenestration operation in January 1946, which brought his hearing to the practical level. Three years after operation he

TABLE 2.—*Audiometric Data on Case 1 (J. F.)*

| | Right Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. | Left Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Date | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | |
| 7/ 8/43 | 15 | 15 | 15 | 25 | 30 | 25 | 15 | 15 | 27 | .... | 45 | 40 | 40 | 55 | 55 | 55 | 40 | 50 | 53 | .... |
| 7/ 9/43 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... | Fenestration | | | | | | | | | |
| 7/10/44 | 15 | 15 | 20 | 20 | 30 | 30 | 20 | 25 | 26 | +1 | 15 | 15 | 20 | 20 | 30 | 35 | 30 | 45 | 28 | +27 |
| 8/16/45 | 15 | 10 | 10 | 25 | 20 | 15 | 10 | 15 | 20 | +7 | 20 | 20 | 20 | 20 | 25 | 40 | 35 | 50 | 28 | +27 |
| 2/ 7/47 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... | Roaring, dizziness, fulness, drop in hearing for 2 months | | | | | | | | | |
| 3/29/48 | 25 | 15 | 10 | 30 | 30 | 25 | 20 | 10 | 28 | —1 | 25 | 15 | 15 | 25 | 45 | 50 | 45 | 50 | 40 | +15 |
| 3/29/48 | Symptoms all relieved after histamine (0.1 cc. 1:100,000,000 dilution) 4 injections | | | | | | | | | | | | | | | | | | | |
| | 30 | 15 | 20 | 40 | 40 | 15 | 15 | 10 | 31 | —4 | 25 | 15 | 25 | 30 | 20 | 30 | 30 | 60 | 30 | +23 |
| 8/19/49 | .. | 25 | 20 | 40 | 40 | 25 | 10 | 25 | 35 | —8 | .. | 30 | 20 | 25 | 20 | 25 | 25 | 65 | 27 | +28 |

TABLE 3.—*Data on Case 2 (D. F.)*

| | Right Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. | Left Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Date | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | |
| 1/ 7/46 | 55 | 55 | 55 | 65 | 65 | 60 | 50 | 60 | 63 | .... | 50 | 45 | 45 | 50 | 65 | 60 | 45 | 45 | 58 | .... |
| 1/10/46 | Fenestration | | | | | | | | | | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... |
| 5/22/47 | 15 | 10 | 5 | 20 | 25 | 25 | 40 | 65 | 23 | +40 | 45 | 40 | 45 | 45 | 60 | 50 | 55 | 55 | 52 | +6 |
| 12/ 2/48 | .. | 0 | 0 | 10 | 25 | 30 | 40 | 80 | 22 | +41 | .. | 55 | 35 | 45 | 45 | 53 | 60 | 80 | 48 | +10 |
| 3/21/49 | Distortion of tones, drop in hearing, fulness off and on for 3 months | | | | | | | | | | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... |
| | .. | 45 | 55 | 65 | 55 | 40 | 55 | 70 | 53 | +10 | .. | 40 | 45 | 50 | 75 | 65 | 55 | 65 | 63 | —5 |
| | Histamine (0.1 cc. 1:10,000,000 dilution) and salt-free diet | | | | | | | | | | | | | | | | | | | |
| 4/14/49 | .. | 5 | 5 | 15 | 30 | 30 | 35 | 75 | 25 | +38 | .. | 40 | 45 | 50 | 60 | 60 | 50 | 60 | 57 | +1 |

experienced an intermittent distortion of tones in the ear operated on, with a sense of fulness and a precipitous drop in hearing. This condition continued intermittently for three months. An injection of 0.1 cc. of 100,000,000 histamine phosphate had no effect on the symptoms. A second injection, a week later, of 0.1 cc. of a 1:10,000,000 dilution of histamine phosphate, with 2 drops of a 1:100,000 dilution by mouth twice daily resulted in return of the hearing to the practical level that same evening, where it has remained for the past six months.

CASE 3.—M. O'C., a woman aged 54, had a fenestration operation in August 1941, which brought her hearing from the 62 decibel level to the 40 decibel level, where it remained for five and one-half years. In December 1946 her hearing began to decline, associated with a sense of fulness in the ear. In January 1947 she was given a 1:100,000,000 dilution of histamine phosphate, 0.1 cc. with 2

drops of a 1:10,000,000 dilution by mouth twice daily. After the first two injections the patient was no better, and nicotinic acid by mouth sufficient to cause flushing did not help, but after the third injection of histamine her hearing returned to the 40 decibel level, where it remained for a year. A second drop in hearing associated with a sense of fulness in the ear operated on was not improved with administration of a 1:100,000,000 or a 1:1,000,000,000 dilution of histamine phosphate, but when she was placed on a salt-free diet with potassium chloride, her hearing returned to the 40 decibel level.

TABLE 4.—*Data on Case 3 (M. O'C.)*

| | Right Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. | Left Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Date | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | |
| 8/29/41 | 60 | 60 | 60 | 65 | 60 | 50 | 45 | 60 | 58 | .... | 75 | 60 | 65 | 70 | 60 | 55 | 60 | 65 | 62 | .... |
| 9/30/41 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... | Fenestration | | | | | | | | | |
| 9/18/42 | 50 | 55 | 55 | 60 | 65 | 50 | 50 | 40 | 58 | 0 | 30 | 35 | 35 | 50 | 45 | 25 | 50 | 40 | 40 | +22 |
| 12/19/47 | 50 | 40 | 45 | 55 | 55 | 55 | 50 | 45 | 55 | +3 | 45 | 30 | 35 | 45 | 35 | 30 | 35 | 45 | 40 | +22 |
| 3/10/49 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... | Plugged sensation with pain, pressure and drop in hearing for 3 weeks | | | | | | | | | |
| | .. | 55 | 60 | 65 | 60 | 65 | 65 | 70 | 63 | −5 | .. | 50 | 55 | 70 | 60 | 60 | 60 | 55 | 63 | −1 |
| 4/28/49 | Symptoms temporarily improved by histamine (0.1 cc. 1:1,000,000,000 dilution); patient better with salt-free diet and potassium chloride | | | | | | | | | | | | | | | | | | | |
| | .. | 55 | 65 | 70 | 70 | 65 | 65 | 70 | 68 | −10 | .. | 40 | 45 | 50 | 55 | 55 | 55 | 55 | 60 | +22 |

TABLE 5.—*Data on Case 4 (H. L. T.)*

| | Right Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. | Left Ear | | | | | | | | Average for: 512, 1024, 2048 | Postoperative change in db. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Date | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | | 64 | 128 | 256 | 512 | 1024 | 2048 | 4096 | 8192 | | |
| 1/24/44 | 80 | 40 | 60 | 45 | 55 | 25 | 95 | 35 | 42 | .... | 40 | 40 | 45 | 45 | 60 | 35 | 95 | 35 | 46 | .... |
| 1/27/44 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... | Fenestration | | | | | | | | | |
| 3/ 5/45 | 50 | 50 | 50 | 55 | 55 | 45 | 90 | 55 | 51 | −9 | 30 | 30 | 15 | 15 | 25 | 30 | .. | 65 | 23 | +23 |
| 9/ 8/45 | .. | .. | .. | .. | .. | .. | .. | .. | .. | .... | Drop in hearing; fullness and tinnitus for 3 months | | | | | | | | | |
| | 45 | 40 | 45 | 55 | 40 | 35 | 95 | 55 | 43 | −1 | 50 | 40 | 55 | 60 | 55 | 40 | 95 | 70 | 51 | −5 |
| 10/ 8/45 | Histamine (0.05 cc. 1:10,000,000 dilution) 2 injections | | | | | | | | | | | | | | | | | | | |
| | 40 | 40 | 40 | 60 | 30 | 25 | 85 | 50 | 32 | +10 | 30 | 25 | 20 | 20 | 15 | 30 | 85 | 60 | 21 | +25 |
| 8/22/46 | 55 | 60 | 40 | 50 | 50 | 40 | 90 | 60 | 47 | −3 | 35 | 25 | 25 | 25 | 30 | 30 | 80 | 55 | 28 | +18 |
| 1/14/47 | Hearing down again, not helped by histamine (0.1 cc. 1:10,000,000 dilution) | | | | | | | | | | | | | | | | | | | |
| | .. | 40 | 40 | 45 | 50 | 35 | .. | 60 | 43 | −4 | 50 | 50 | 55 | 65 | 50 | 15 | 90 | 65 | 53 | −7 |
| 2/27/47 | Histamine (0.1 cc. 1:1,000,000 dilution) with 2 drops (1:100,000 dilution) per os twice daily | | | | | | | | | | | | | | | | | | | |
| | 45 | 40 | 45 | 50 | 45 | 35 | 90 | 55 | 43 | −1 | 20 | 20 | 20 | 20 | 30 | 45 | 80 | 60 | 27 | +19 |
| 6/24/47 | Hearing good as long as histamine continued (10 drops 1:10,000 dilution) per os twice daily | | | | | | | | | | | | | | | | | | | |
| | 50 | 40 | 45 | 50 | 45 | 40 | 85 | 55 | 45 | −3 | 15 | 15 | 15 | 15 | 30 | 25 | 75 | 65 | 23 | +23 |
| 12/10/48 | Histamine no longer of aid; slight dizzy spell | | | | | | | | | | | | | | | | | | | |
| | 45 | 45 | 50 | 55 | 45 | 35 | 75 | 55 | 44 | −2 | .. | 55 | 60 | 65 | 70 | 55 | 85 | 55 | 63 | −17 |

CASE 4.—H. L. T., a man aged 37, had a fenestration operation in June 1943, which brought his hearing to the practical level, where it remained a year and a half. In July 1945 his hearing began to decline associated with a sense of fulness in the ear and an increase in tinnitus. In September the patient was given two injections two weeks apart of 0.05 cc. of a 1:10,000,000 dilution of histamine phosphate subcutaneously. His hearing slowly returned from a 51 decibel loss to the practical level. Since that time, that is, for the past four years, his hearing has

continued to fluctuate markedly. The use of alcohol and tobacco definitely makes it worse, and histamine sometimes but not always improves it, the beneficial response to dilute histamine solution becoming gradually less.

## COMMENT

A syndrome consisting of dizziness, tinnitus, a drop in hearing, a sense of fulness and sometimes pain in the ear operated on may occur at any time after the fenestration operation, one or more of these symptoms being present at the same time in any one case, rarely all five at once. This syndrome closely resembles Ménière's symptom complex and is probably due to a similar mechanism, namely, a labyrinthine hydrops. Hilger has recently suggested that the basic cause for labyrinthine hydrops is an imbalance in the autonomic innervation of the arterioles to the inner ear, resulting in periodic arteriole vasospasm with peripheral capillary dilatation and exudation. The exact relation of histamine to this vasospasm is not clear, but the fact that this labyrinthine syndrome occurs months or years after local surgical trauma to the labyrinth suggests a possible local sensitization of the tissues to histamine as the result of the local liberation of this substance at the time of operation. Perhaps treatment with minute doses of histamine desensitizes the tissues and thus relieves the symptoms. Some patients not relieved by histamine alone are relieved by a salt-free diet with potassium chloride, alone or in conjunction with the histamine. Antihistaminic drugs, such as tripelennamine hydrochloride (pyribenzamine hydrochloride®), have not been effective in the few cases in which they were used.

## CONCLUSIONS

A syndrome of dizziness, tinnitus, a sense of fulness in the ear, a depression in hearing and pain occurs not infrequently in the ear operated on several months or years after a fenestration operation.

This syndrome closely resembles and probably is a labyrinthine hydrops.

The administration of dilute histamine solution sometimes, but not always, relieves the symptoms.

The mechanism of the beneficial effect of therapy with dilute histamine solution is not known. It is suggested that perhaps a local sensitization of the otic labyrinth to histamine may cause the symptoms and that injections of minute doses of histamine may serve to desensitize the labyrinth.

55 East Washington Street.

# USE OF SPEECH TESTS FOR EVALUATION OF CLINICAL PROCEDURES

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ST. LOUIS

IN THE past decade interest in clinical auditory tests has been greatly stimulated by a wide, although not wholly unrelated, variety of factors. The more significant among these factors are the development of electroacoustic instruments, which make possible increasingly refined measurements; growing interest in the fenestration operation; expanding consumer acceptance of improved hearing aids; evolution of promising technics of research in psychoacoustics, and, finally, but none the less stimulating, the encouraging cooperative attack by workers from allied fields on the problems presented by aural rehabilitation.

From the standpoint of clinical technics employed, auditory tests fall into three broad categories: (1) tuning fork, (2) pure tone audiometry and (3) articulation tests.[1] Although the evolution of these tests overlap somewhat in time, the order in which they are presented represents basically the sequence of their refined development. The basic principles of articulation testing have not been implemented universally in routine otologic practice, probably because of the recency of their emergence, the lack of standardization of operating technics and the inadequacy of instrumentation. It is the purpose of this paper, therefore, to deal with the principles underlying articulation tests and to suggest how the results of the tests may be applied to clinical situations.

## ARTICULATION FUNCTION

The articulation function is the basic concept involved in articulation testing. Essentially, it expresses the relation between the percentage of speech units (syllables, words, sentences) heard correctly and the inten-

From the Central Institute for the Deaf.

This paper is the first in a "Symposium on Pure Tone and Speech Testing," held at the American Academy of Ophthalmology and Otolaryngology, Chicago, on Oct. 8, 1949.

This paper is an adaptation, with revisions, of an address I delivered to the Section of Otology of the Royal Society of Medicine, London, May 6, 1949, and is to be published in the Proceedings of the Royal Society of Medicine.

1. Covering promising tests with use of conditioning and electrical technics for young children suspected of deafness is beyond the scope of this paper.

sity of the speech at the ear of the listener. The notion of the articulation function grew out of the attempts by Fletcher and Steinberg [2] and their associates at the Bell Telephone Laboratories to devise methods of assessing the efficiency of telephone systems in transmitting speech. The use of speech as the test material was logical, since the purpose of the telephone, is obviously, to transmit speech. An example of one of

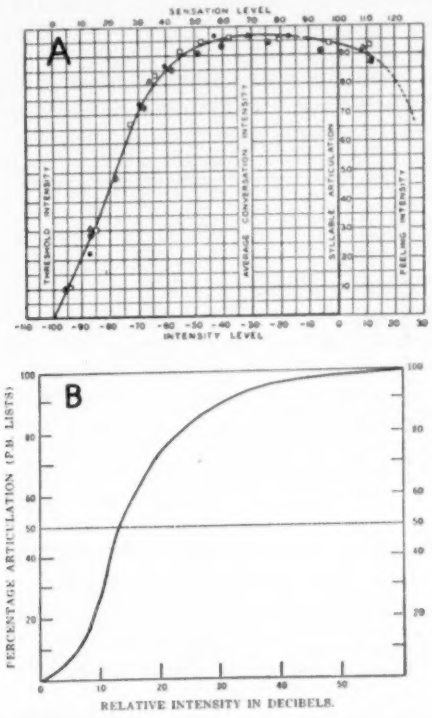


Fig. 1.—*A*, articulation versus intensity of received speech.[3] *B*, articulation function for normal ear showing increase in percentage of words repeated correctly with increase in intensity.[4]

Fletcher's [3] early articulation functions is shown in figure 1*A*. It is seen that as the speech becomes more intense the listener hears more syllables

2. Fletcher, H., and Steinberg, J. C.: Articulation Testing Methods, Bell Syst. Tech. J. **8**:4, 1929.

3. Fletcher, H.: Speech and Hearing, New York, D. Van Nostrand Company, 1929.

correctly. A simpler form of articulation function is shown in *B* of the same figure.[4] The ordinate shows the articulation score in percentage of words repeated correctly, and the abscissa gives the relative intensity in decibels. In this case, a list of 50 phonetically balanced words (recognized as proportionately representative of the phonetic elements of English as it is normally spoken) is presented to the listener at enough levels, 10 decibels apart, to complete the function. A subject, therefore, is credited with 2 per cent on the vertical axis for each word repeated correctly. Note also the manner in which the articulation score climbs as a function of intensity—especially the steepness of the slope and the 30 to 40 decibel range over which almost maximum articulation is achieved.

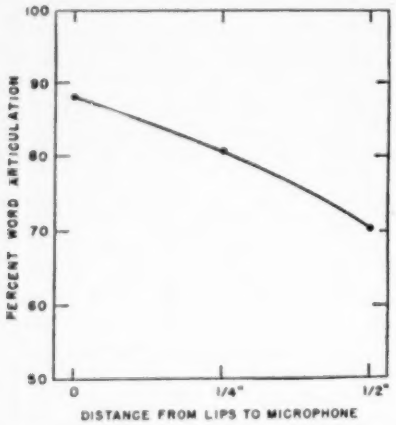


Fig. 2.—Graph showing the effect on communication of varying the distance of the microphone from the lips. The test was made with the announcer and crew of listeners in an atmosphere of airplane noise (115 decibels). Each point is based on the average of tests read by three announcers.[5]

It is apparent that articulation scores (and functions) are subject to the influence of many variables, and care should be taken to maintain conditions as constant as possible in making tests, except, of course, for the item being tested, whether it is a communication system or an ear suspected of hearing impairment. In figure 2, from Miller,[5] is seen, for

4. Articulation Testing Methods Report, no. 3802, Emergency Management Office, Office of Scientific Research and Development, November 1944, pt. 2.

5. Miller, G. A.: Transmission and Reception of Sounds Under Combat Conditions, Summary Technical Report, National Defense Research Committee, Washington, D. C., 1946, vol. 3, chap. 5, div. 17.

example, how distance from lips to microphone may influence the percentage of word articulation.

### DETERMINATION OF THE THRESHOLD OF HEARING BY ARTICULATION TESTS

It is well at the outset to clarify the concept of the threshold as it is derived from articulation tests. Three thresholds can be distinguished: (1) the threshold of detectability, defined as the point at which the listener is just able to detect speech sounds about half the time and is not able ordinarily to identify any of the sounds themselves; (2) the threshold of perceptibility, defined as the point at which the listener begins to perceive some words but can barely follow the gist of connected speech, and (3) the threshold of intelligibility, defined as the point at which the listener understands half the material presented to him and can presumably follow without perceptible effort the gist of connected speech. For the purposes in the clinical context only the threshold of intelligibility will be needed.

In constructing a test to determine the threshold of intelligibility, Hudgins and associates[6] suggested that the test items should meet the following criteria: (1) familiarity—the vocabulary should be within the intellectual ken of the listener; (2) phonetic dissimilarity—fine discriminations (cowboy, plowboy) should not be necessary, since no useful purpose is served by them in threshold tests; (3) normal sampling of English speech sounds—not essential for threshold testing, but a reasonable sampling is desirable, and (4) homogeneity with respect to basic audibility—the ease with which test words are understood should be as equivalent as possible so that small numbers of items can be scrambled and the articulation function will rise steeply over a narrow range of intensity.

A test which meets these criteria has been developed by Hudgins and associates at the Psycho-Acoustic Laboratory of Harvard University. It consists of two lists of 42 dissyllabic words of the spondee stress pattern, i. e., such words as earthquake, hardware, in which both syllables are accented equally. The subject hears 6 words at given levels progressively attenuated in 4 decibel steps. The point at which he repeats correctly 3 (half the words) is the threshold of hearing (by the previous definition). From the standpoint of hearing impairment, the hearing loss can be defined as the number of decibels more required by the impaired ear to hear the equivalent number of words (3). Figure 3*A* shows the hearing losses of two ears of different subjects.

6. Hudgins, C. V.; Hawkins, J. E.; Karlin, J. E., and Stevens, S. S.: The Development of Record Auditory Tests for Measuring Hearing Loss for Speech, Laryngoscope **57**:57-89, 1947.

Subject *A* has a hearing loss of 14 decibels, and subject *B* has a loss of 55 decibels. In other words, subject *A* requires 14 and subject *B* requires 55 decibels more than one with a normal ear to hear the same number

A sentence test (no. 12) based on the same principles also has been developed by the Psycho-Acoustic Laboratory.

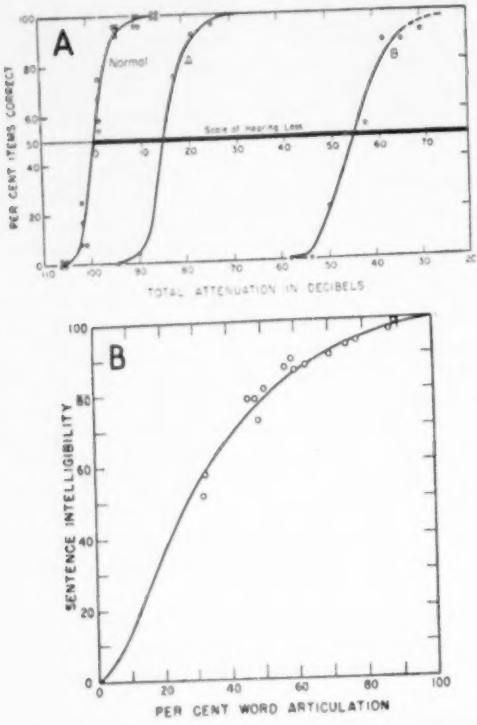


Fig. 3.—*A*, graph showing how the hearing loss for each of 2 subjects, *A* and *B*, is established in terms of the articulation function obtained from a group of listeners with normal hearing. Hearing loss is equivalent to the horizontal distance (in decibels) between the curve for the normal and that for the defective ear. Since the threshold is based on the normal score of 50 per cent accuracy, the horizontal line through the middle of the plot provides a scale of hearing loss.[6] *B*, results of comparison of lists of words and lists of sentences for intelligibility under a wide variety of conditions. Each point on the graph represents one test of 100 words and one test of 50 sentences. The curve passing through the data was derived from results obtained at the Bell Telephone Laboratories.[7]

The relation of per cent word articulation and sentence intelligibility depends, of course, on the type of words and sentences used. A fairly

typical curve of this relation plotted by Egan [7] is given in figure 3*B*. Hudgins and associates [8] found that the threshold for the no. 12 (sentence intelligibility) test is about 4 decibels above the threshold for no. 9 (spondees).

Another speech test (not using the concept of the articulation function) has been described by Falconer and Davis.[8] In this test the subject listens to a sample of recorded connected discourse and manipulates the attenuator until he is just able to get the gist of what is being said. His hearing loss is the number of decibels more which his ear requires than the normal ear to reach his threshold of intelligibility for connected discourse. This test has the advantage of economy of time and high face validity. The end point, on the other hand, is relatively not too definitive. However, Thurlow and associates [9] found that hearing losses for speech determined by a variety of the types of tests described correlated so closely with one another that only one of them need be used in any clinical routine.

### TESTING AUDITORY DISCRIMINATION

It is important not only to determine the faintest speech which an ear can hear (measured by threshold type tests described in previous section) but it is of great clinical significance (as I shall point out later) to know how the ear hears speech at any intensity, particularly that above threshold. Clinicians are familiar with the patient who says "I hear but I can't understand; I can hear better if you speak clearly rather than loudly." This patient's problem is not clearly delineated by the threshold test. Granted that a sound needs to be audible before it can be distinguished from the other sounds, the fact that it is audible does not necessarily guarantee that it can be recognized accurately. The ability to distinguish one sound from another when both are audible is called discrimination as distinguished from sensitivity, which refers to the faintest sound which the ear can hear. In addition to sensitivity, a high degree of discrimination is needed to discriminate sin from thin and pit from pith.

In constructing a test to measure the power of discrimination (or, clinically, the discrimination loss) Egan [7] suggested consideration of these points: (1) representation of fundamental speech sounds—reasonable proportional representation in the test lists of the sounds that occur in

7. Egan, J. P.: Articulation Testing Methods, Laryngoscope **58**:955-991, 1948.

8. Falconer, G. A., and Davis, H.: The Intelligibility of Connected Discourse as a Test for the "Threshold of Speech," Laryngoscope **57**:581-595, 1947.

9. Thurlow, W. R.; Silverman, S. R.; Davis, H., and Walsh, T. E.: A Statistical Study of Auditory Tests in Relation to the Fenestration Operation, Laryngoscope **58**:43-66, 1948.

everyday speech insures that the test measures what it sets out to measure, i. e., how the ear copes with the task of discriminating speech which it is likely to have to discriminate in routine oral communication; (2) types of test items—words appear to be preferable to nonsense syllables, which may require recording by phonetic symbols and to sentences which may afford contextual clues, and (3) difficulty and reliability of test lists—the test items must be so selected that the distribution of item difficulty in each list will make possible a sensitive

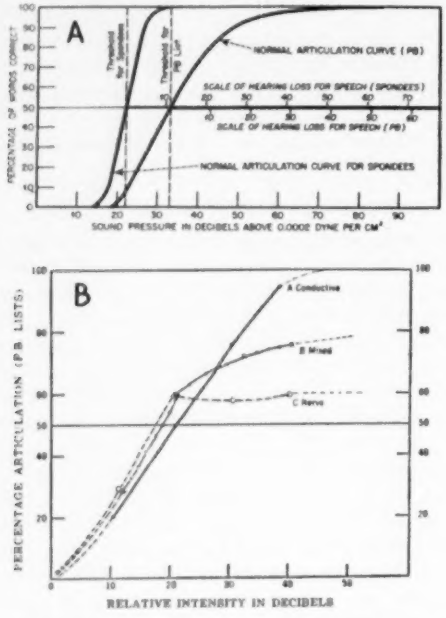
A
NORMAL ARTICULATION CURVE (PB)
SCALE OF HEARING LOSS FOR SPEECH (SPONDEES)
SCALE OF HEARING LOSS FOR SPEECH (PB)
NORMAL ARTICULATION CURVE FOR SPONDEES
PERCENTAGE OF WORDS CORRECT
SOUND PRESSURE IN DECIBELS ABOVE 0.0002 DYNE PER CM²
B
A Conductive
B Mixed
C Nerve
PERCENTAGE ARTICULATION (PB LISTS)
RELATIVE INTENSITY IN DECIBELS

Fig. 4.—*A*, graph showing difference in slopes of two types of articulation curves.[10] *B*, graph showing articulation curves for conduction, mixed and nerve deafness.[11]

measuring instrument. In other words, those items which under conditions of the tests are always recorded correctly or are always missed should be eliminated from the test lists. Of course, for both sensitivity tests and discrimination tests many equivalent lists should be available to cut down the learning factor when more than one test is necessary.

Egan[7] and his associates at the Psycho-Acoustic Laboratory constructed a test which consists of 20 equivalent phonetically balanced lists of 50 words each. Figure 1 *B* shows the articulation function which is

derived from the phonetically balanced lists. The discrimination loss, measured along the vertical axis, would be the percentage of words that the ear could not discriminate at an intensity level sufficiently high above threshold to eliminate the factor of questionable sensitivity. In other words, no matter how loud the speech is made, the ear may still fail to discriminate all the test words.

A comparison of the shapes of the no. 9 test and phonetically balanced curves taken from Davis [10] is shown in figure 4 *A*. Notice that the spondee curve rises steeply because the words are almost equally audible and intelligible, whereas the phonetically balanced curve rises more gradually because the words represent the broad range of difficulty encountered in everyday speech.

## CLINICAL APPLICATIONS OF ARTICULATION TESTS

Articulation tests, properly applied, serve a useful purpose for the clinician who is confronted routinely with problems of diagnosis, prognosis and evaluation of medical and surgical procedures and hearing aids.

*Diagnosis and Prognosis.*—The otologist is called on routinely to differentiate conduction and nerve deafness. Or, otherwise stated, he must assess cochlear function. The patient who is a candidate for fenestration surgery is a case in point. The patient can be tested with the phonetically balanced lists at high intensity levels (free from distortion), and if his articulation score increases proportionately with intensity, he is likely to have adequate cochlear function. In essence, an attempt is made to accomplish prior to operation what the operation sets out to do, namely, to deliver sound to the analyzing mechanism in the inner ear by overriding or by-passing a conduction lesion. If the attempt to deliver sound over the electroacoustic system is successful, as judged by the maximum articulation score, then surgical procedure, intended to alter beneficially the transmission mechanism of the ear, should accomplish the same result.

Figure 4 *B* shows the unsmoothed articulation functions taken from the clinical records of Walsh and Silverman.[11] Curve *A* is that of a patient who reaches a relatively high maximum articulation score if he is presented with speech at high intensity. This patient, therefore, has adequate cochlear reserve and would be a good candidate for operation. Curve *B* represents a patient with mixed deafness, since his articulation score does not rise with intensity in the same manner as curve *A*. The conduction aspect of his deafness might be helped by fenestration, but his prognosis would be less favorable than that of the first patient. Note

10. Davis, H.: Hearing and Deafness: A Guide for Laymen, New York, Rhinehart & Co., Inc., 1947, chap. 6.

11. Walsh, T. E., and Silverman, S. R.: Diagnosis and Evaluation of Fenestration, Laryngoscope **56**:536-555, 1946.

that in curve *C* increasing the intensity does not proportionately elevate the articulation score after a certain maximum has been reached. The curve of this patient shows that adequate cochlear function is not present. Operation might provide for more sound to be delivered to the inner ear, but lack of adequate cochlear reserve would preclude reception beneficial to the patient. Walsh and Silverman pointed out further that the thresholds of the three curves are practically identical, but the diagnosis and prognosis are based on auditory function above threshold; consequently, prime significance is attached to the shape of the articulation function and particularly to the maximum articulation score. The test would have obvious limitations in certain special types of perceptive deafness, which, if suspected, must be explored carefully. For example, Dix, Hallpike and Hood [12] suggested that the maximum articulation score might differ in cases of end organ lesions and in nerve fiber lesions.

*Evaluation and the Concept of Social Adequacy.*—In order practically to evaluate therapeutic procedures and hearing aids one generally needs to know to what extent one has improved the patient's ability to cope with the everyday dynamics of oral communication.

To this end, Walsh and Silverman [11] suggested the concept of the social adequacy index for hearing, which essentially is the average percentage of phonetically balanced words that would be correctly understood by the patient at the faint, the conversational and the loud speech levels. In other words, just as one is accustomed to averaging hearing loss at critical frequencies, one averages ability to hear speech at critical intensities, in this case determined by social criteria. The threshold measure is not in itself adequate to predict a patient's performance, since it is seen that ability to discriminate speech can vary widely above threshold in patients who may have the same threshold scores. The social adequacy index, on the other hand, evaluates the remedial measures in terms of shifts both in sensitivity and in discrimination. It states, for example, that the patient with poor discrimination is not helped by others' shouting, as one must do in a noisy place, because an increase in intensity does not improve his power of discrimination. In a sense, the social adequacy index accounts for hearing performance in a dynamic acoustic environment. Of course, in cases in which the discrimination is not expected to change perceptibly the shift in threshold will be adequate for evaluation. In figure 5 *A* are shown the articulation functions of an ear before and after the fenestration operation related to the critical threshold of adequacy and to the curve for the normal ear.

12. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: "Nerve" Deafness: Its Clinical Criteria, Old and New, Proc. Roy. Soc. Med. **42**:527-536 and 540, 1949.

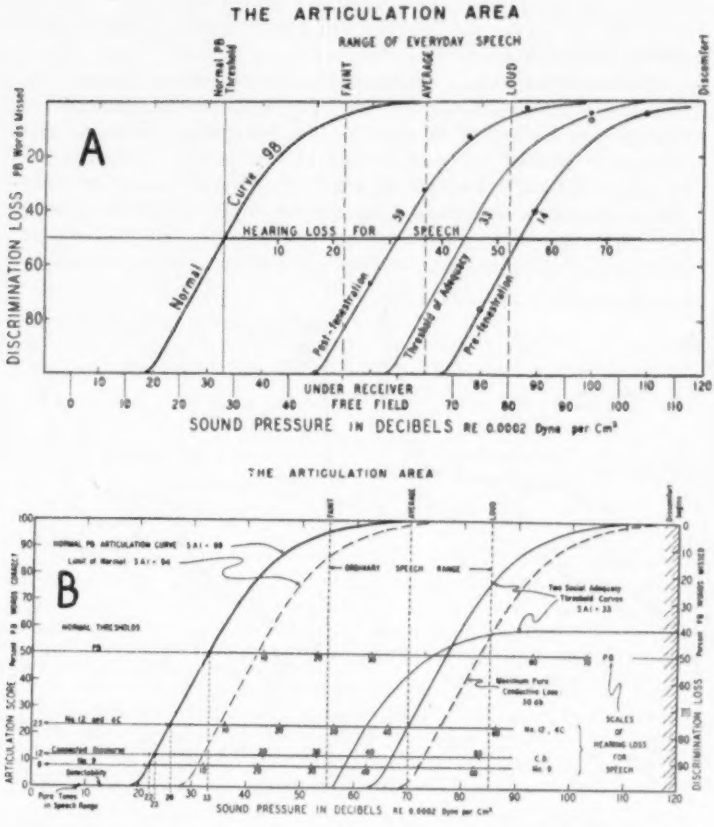


Fig. 5.—*A*, graph showing change in articulation function in fenestrated ear. *B*, version 1, made on Feb. 1, 1948. These curves are based on the phonetically balanced word lists of the Psycho-Acoustic Laboratory, as recorded at the Central Institute for the Deaf, as of January 1948. The other thresholds are for recorded tests used with the same apparatus: no. 9 (spondee words) and no. 12 (sentences), Psycho-Acoustic Laboratory, Harvard; 4C (2 digit numbers), Western Electric Company, and connected discourses (thresholds of intelligibility and detectability), newscast by Fulton Lewis Jr., Central Institute for the Deaf. The social adequacy index is the average percentage of phonetically balanced words that would be correctly understood by the patient at the three speech levels indicated by the dotted lines.

Davis [13] elaborated the notion of the social adequacy index and brought into quantitative relation the various articulation tests. This is shown in figure 5 *B*. He also presented a table for the rapid calculation of the social adequacy index.

Frequently, clinicians evaluate measures to improve hearing by soliciting a subjective judgment from the patient on the status of his hearing. In a series of 161 patients with fenestration, Silverman and associates [14] studied the relation between the patients' judgments of the status of their hearing and the results of articulation tests performed under laboratory conditions. The patients were furnished a rating scale for the various conditions under which they had to listen in everyday life (noisy places, audience situations, group conversation,

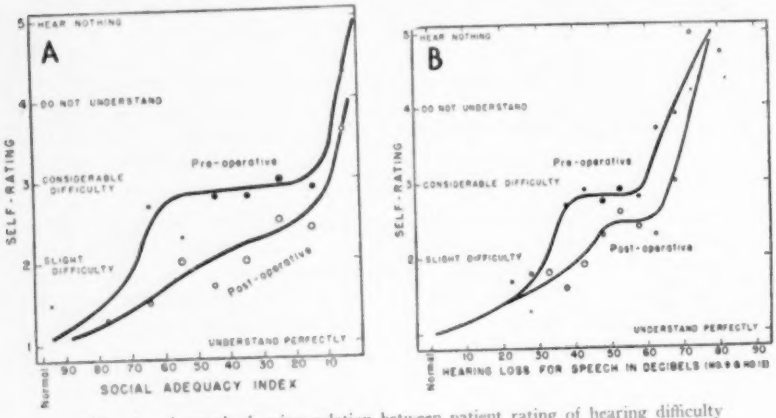


Fig. 6.—*A*, graph showing relation between patient rating of hearing difficulty and the social adequacy index, preoperative and postoperative. For some unexplained reason the patients seem to rate themselves as hearing more "accurately" after the operation.[14] *B*, graph showing relation between patient rating of hearing difficulty and hearing loss for speech, preoperative and postoperative.[14]

etc.). Figure 6 shows the relation between patient judgments, actual measured losses and social adequacy index scores. Theoretically, one should expect linear relationships, i. e., the greater the hearing loss (or the lower the social adequacy index) the greater difficulty in hearing, as judged by the patient. The plateau in both curves indicates that this situation is not so, probably because it is possible to compensate by the

13. Davis, H.: The Articulation Area and the Social Adequacy Index for Hearing, Laryngoscope **58**:761-778, 1948.

14. Silverman, S. R.; Thurlow, W. R.; Davis, H., and Walsh, T. E.: Improvement in the Social Adequacy Index for Hearing Following the Fenestration Operation, Laryngoscope **58**:607-631, 1948.

raised voice for losses of hearing up to 60 decibels. Note, too, the changes in judgments postoperatively. At any rate, patient judgment as a technic of evaluation should be used with extreme caution. The table, from Silverman and associates,[14] indicates the numbers in terms of hearing loss and social adequacy index, which may be placed on certain critical social thresholds. Incidentally, this information may be useful to those who must deal with job classification and medicolegal problems.

EQUIPMENT

It is likely that simple, inexpensive assemblies of standardized equipment soon will be available and within reach of the practicing otologist, but even when this comes to pass precautions concerning characteristics and calibration must be observed. After all, articulation scores are

*Thresholds of Hearing*

| Threshold | Loss for Speech Decibels | Social Adequacy Index |
|---|---|---|
| Normal | 0 | 98 |
| Limit of normal | 9 | 94 |
| Social difficulty begins | 30 | 67 |
| Threshold of social adequacy | 45 | 33 |
| Limit of compensation | 60 | 10 |
| Limit of compensation with hearing aid | (To be determined) | |

relative scores contingent on a variety of factors. Only relative statements can be made about them within a framework of accurately described equipment and conditions.

Finally, work in the field of articulation testing, at least in the present state of the art, should be viewed critically and, in some instances, experimentally. Ongoing research says that articulation tests need to be increasingly refined and validated, and they need to be further streamlined for wider clinical application. Certainly, this can be said about most clinical technics. Articulation tests are not intended to supplant orthodox clinical tests, but they are a valuable supplement and they should hold a respectable place in the armamentarium of the clinical otologist, because they are quantitative, reproducible, reasonably valid and tend to objectivize the responses of the patient. After all, in articulation testing the patient either repeats the word correctly or he does not. He cannot get by with "maybe."

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# CLINICAL APPLICATION OF BONE CONDUCTION AUDIOMETRY

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EVANSTON, ILL.

MANY otolaryngologists distrust bone conduction audiometry. Attempts to use it as a diagnostic tool in clinical practice have frequently been unsatisfactory and discouraging. It is true that some otologists have come to rely on the technic, but they have encountered difficulty when comparing results. Generally speaking, bone conduction audiometry is considered a relatively uncertain method which serves merely as a diagnostic supplement to tuning fork tests.

## DIFFICULTIES IN BONE CONDUCTION TESTING

Four major reasons contribute to the current mistrust of bone conduction measurements. In the first place, many workers doubt the reliability of thresholds obtained by this method. They feel that a high margin of error is introduced by such factors as differences in positioning of the vibrator and in the pressure with which it is applied. However, Carhart and Hayes have shown that bone conduction thresholds can have good repeatability in clinical circumstances.[1] They compared tests on 500 ears. Statistically, thresholds for bone conduction were found to be superior in repeatability to thresholds for air conduction. It should be noted that all bone measurements were made with oscillators of the hearing aid type held in steel headbands. Positioning and pressure did not emerge as highly disturbing variables when this type of vibrator was used and when the ordinary precautions of clinical audiometry were maintained. In such circumstances, a bone conduction curve may be interpreted with a margin of uncertainty no greater than the margin which must be assigned to the companion air conduction curve.

The second reason for distrust of bone conduction audiometry is more serious. It involves the calibration of audiometers. Manufacturers have disagreed about the values to assign for normal threshold. (To be sure, serious technical obstacles have been responsible. Nevertheless, the fact remains that clearly established standards have not yet been made.) The end result has been confusion when tests made with

This paper is the second in a "Symposium on Pure Tone and Speech Testing" held at the American Academy of Ophthalmology and Otolaryngology, Chicago, on Oct. 8, 1949.

1. Carhart, R. and Hayes, C.: The Clinical Reliability of Bone Conduction Audiometry, Laryngoscope **59**:1084-1101, 1949.

different audiometers have been compared. Furthermore, the situation has been aggravated because manufacturers have not always maintained as rigorous production and service standards for bone units as for air units. It is logical that persons attempting to use bone conduction should lose confidence in the method when faced with the uncertain validity which these factors represent.

The third reason for doubt of the bone conduction technic has arisen from inadequate testing conditions and imperfect office checks on calibration. Again, technical obstacles are numerous, but they are not serious if clearly understood. For one thing, even small amounts of room noise will mask the normal ear while it is being tested by bone conduction. In consequence, false thresholds will be obtained with patients having near-normal hearing. False thresholds will also appear in attempts to calibrate the bone unit with normal ears. Faulty calibration leads to incorrect positioning of the bone conduction curve on the audiogram chart. Whenever the error is large, the resultant air-bone relation will be illogical in view of other diagnostic criteria. Quite naturally, the bone measurements are disregarded and the technic is distrusted.

Finally, bone conduction audiometry places special emphasis on controlled masking of the opposite ear. Unfortunately, the clinical application of controlled masking has been shrouded in confusion. Its practical use requires a calibrated noise whose effective masking is known. Most audiometers and special masking units have not been supplied with a noise which is so calibrated. Usually, therefore, the busy physician has been forced to work with an unknown—and frequently inadequate—quantity when he has attempted to apply controlled masking in his pure tone audiometry. His bone conduction measurements have suffered most because masking of the opposite ear is oftener critical in this kind of testing and because of threshold shifts which occur when some types of ear are occluded by the masking headphone.

## REQUIREMENTS FOR ADEQUATE TESTING

The limitations just reviewed are not insurmountable. Adequate bone conduction audiometry has four special requirements.

In the first place, the kind of audiometry room which is acceptable depends on the type of hearing one plans to test. If one wishes accurate bone thresholds on normal and near-normal listeners, a highly soundproof chamber is essential. However, one may have to be satisfied with a room which, though relatively quiet, is not fully soundproof. Here adequate tests with bone conduction are impossible on persons with normal hearing, but one can still obtain useful measurements on hard of hearing patients. Stated differently, any room which is adequate for air conduction testing of normal hearing is also adequate for testing the bone conduction of patients whose hearing loss is

sufficient to constitute a social handicap. For example, good bone measurements may be made on patients whose losses are severe enough to warrant consideration of fenestration surgery. Their hearing losses serve as protection against masking by mild ambient noise. Good testing by bone requires that one remember the limitations which a particular audiometry room imposes and that one restrict accordingly the type of patient given bone tests.

The second requirement is that the audiometer itself shall be an approved instrument which is functioning satisfactorily. General performance can be assured by checking it in the office and by returning the instrument periodically to the manufacturer for service. However, the user must at present make his own empiric calibration of its bone conduction system. Such calibration is easily accomplished. It requires a moderately good testing room and involves three steps.

1. The air conduction circuit is first calibrated, over the range of 256 to 4096 cycles per second, on 10 or 12 young adults whose hearing is known to be normal. The average reading for this group is taken as the norm at each frequency.

2. Next, 10 to 12 ears [2] with pure perceptive losses of intermediate severity are tested by both air and bone conduction. Theoretically, such patients should yield superimposed air and bone curves. If the curves do not superimpose, appropriate corrections should be made in the values assigned to the bone readings. The method for making such corrections is illustrated in figure 1.

(*a*) The average air conduction curve for the group with perceptive losses is first plotted. (Audiometer readings are naturally corrected where necessary in order to conform to the calibration for air obtained in step 1.)

(*b*) Next, the averages of the audiometer readings for bone are plotted on the same chart.

(*c*) The amount, at each frequency, by which the two curves fail to agree represents the correction which must be applied to bone readings for that frequency.

3. The third step is computation from these corrections of the audiometer settings for bone which correspond to normal threshold. This calibration is made to the nearest 5 decibels.

Once the calibration just described has been completed, clinically acceptable measurements of bone conduction threshold may be obtained. The audiometrist merely corrects subsequent audiometric readings for bone by the amounts which the calibration data require. The procedure is simplified by pasting a correction sheet on the face of the audiometer. The audiometrist, when testing a patient, can then glance at the sheet and record the corrected threshold rather than the instrument reading. The resultant audiometric chart will show air-bone relations which are more easily interpreted.

The basic advantage of the foregoing method for calibration is that it is not affected by mild room noise. In my experience, after this type

2. Each patient selected should have an approximately equal loss in the two ears. Observation of this precaution helps to avoid complications and confusion. However, different patients need not have identical losses.

of empiric calibration, bone curves are obtained on hard of hearing patients which are credible in the light of other diagnostic criteria.[3]

Unfortunately, time limitations allow only mention of the last two requirements for adequate bone conduction testing.

The third requirement is an effective system for controlled masking of the opposite ear. The fourth requirement is that the audiometrist be well trained and have good insight. The audiometrist must be capable of adapting his testing routines to the patient and to the details of the loss as they emerge during the test.

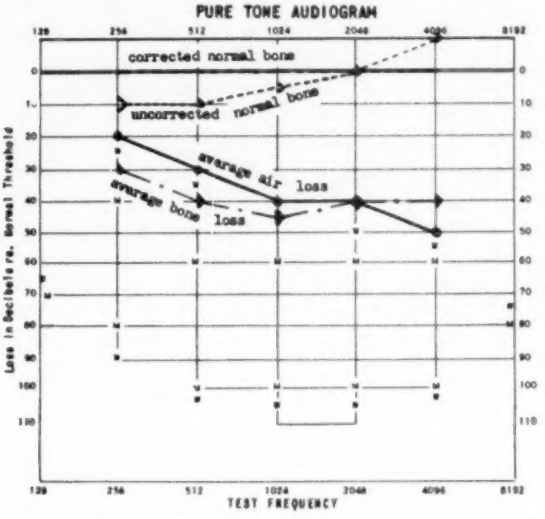


Fig. 1.—Method for calibrating a bone conduction system with a group of patients with mild perceptive losses: (1) Plot average air audiogram for group. (2) Plot average of audiometer readings for bone thresholds. (3) Compute difference between 1 and 2 for each test frequency. (4) Amount of difference is correction which must be applied when patients are tested with the bone system.

## APPLICATION TO DIAGNOSIS

Whenever adequate bone conduction audiometry is available, results can be obtained which are highly useful in diagnosis. Lierle and Reger have stated the basic premises.[4]

3. The reader will note that clinical audiograms offer a continual check on the adequacy of the calibration. Patients with pure perceptive losses should yield records which, within the limits of accuracy of clinical audiometry, show superimposition of the air and bone curves. When such records are not obtained, the calibration is incorrect and the whole procedure of calibration must be repeated.

4. Lierle, D., and Reger, S.: Correlations Between Bone and Air Conduction Acuity Measurements over Wide Frequency Ranges in Different Types of Hearing Impairments, Laryngoscope **56**:187-224, 1946.

I. If the air conduction acuity is below normal but bone conduction normal or possibly increased, the patient has a middle ear lesion.

II. If the patient has the same loss for both air and bone conducted sound, or possibly disproportionately decreased bone conduction, the loss is the result of an inner ear lesion. . . .

III. If the patient has a loss for both air and bone conducted sound, with a greater loss for air than for bone conduction, the impairment is due to a mixed lesion, indicating simultaneous middle and inner ear involvement.[4]

When testing is properly controlled, routine audiograms conform to the foregoing rules within the limits of accuracy one may reasonably

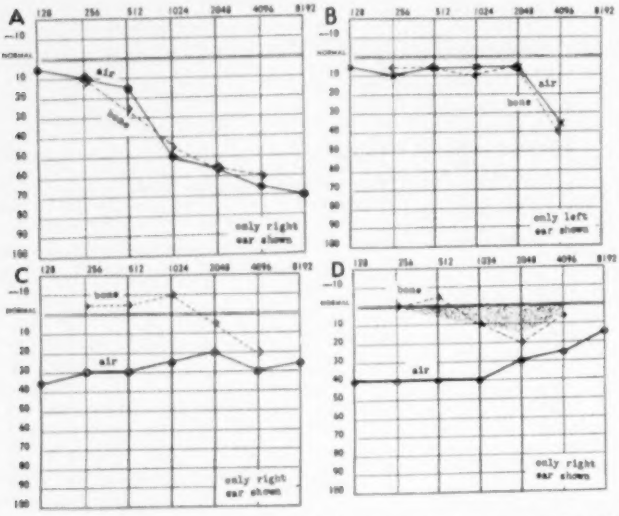


Fig. 2.—*A*, air and bone audiograms for right ear of elderly man with deafness due to presbycusis, illustrating superimposition of curves within limits of accuracy of clinical audiometry. *B*, air and bone audiograms for left ear of young adult with high tone perceptive loss due to acoustic trauma as result of exposure to machine gun fire, illustrating superimposition of curves within limits of accuracy of clinical audiometry. *C*, air and bone audiograms for right ear of teen-aged girl with conduction deafness due to chronic suppurative otitis media; area between the air and bone curves represents the conduction component within the limits of accuracy of clinical audiometry. *D*, air and bone audiograms for right ear of young woman having clinical otosclerosis with incomplete ankylosis of the stapes but without inner ear involvement; shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors.

expect of clinical audiometry. The following examples will illustrate this point.[5]

5. Diagnoses in the cases discussed within this paper were supplied by Dr. George E. Shambaugh Jr., Dr. Arthur Juers and Dr. Paul Campbell.

Figure 2 *A* shows the audiometric record on an ear with nerve deafness diagnosed as due to presbycusis. One is impressed by the manner in which the air and bone curves interweave. Within the limits of accuracy of clinical audiometry, the losses are equal by the two modalities. The case gives no audiometric evidence of a conduction component. This finding was confirmed by all the other diagnostic criteria.

Figure 2 *B* illustrates another type of pure perceptive loss, namely, acoustic trauma due to exposure to machine gun fire. Here, the outstanding feature is the sharp drop in acuity for high frequencies. The air and bone curves again interweave. The loss for bone is the same as the loss for air—as theory implies for a perceptive lesion.

Figure 2 *C* portrays the audiogram yielded by an ear with conduction deafness due to chronic suppurative otitis media. Here, the bone curve is above the normal threshold line except for a drop in the high frequency range, while the air curve is depressed an average of 28 decibels. The gap between the two curves represents the conduction component.

A wide variety of additional examples could be given, but the remainder of this discussion will be limited to some of the audiometric patterns which have been yielded by patients with clinical otosclerosis.

Experience in the Hearing Clinic at the Northwestern University Medical School has revealed a typical audiometric configuration which has frequently been found in cases of clinical otosclerosis without cochlear damage. Figure 2 *D* illustrates this configuration as encountered in a case with incomplete ankylosis of the stapes. One notes two features: First, the air conduction curve rises somewhat at higher frequencies. As will be pointed out later, the slope of the rise appears to be related to the stage in the progression of stapes fixation. Second, the bone conduction contour carries a shallow notch, with its greatest depression at 2048 cycles per second.

The notch in the bone conduction curve is particularly provocative, since it seems to imply a slight component of nerve loss. However, evidence is accumulating which indicates that this notch is the result of purely mechanical factors. In other words, there seems to be a shift in sensitivity to bone stimuli which appears in connection with the process of stapedial fixation. This shift has been observed before fixation is complete. Its details have not been fully defined, but the shaded area in figure 2 *D* represents the estimate on its magnitude, which I have made on the basis of the clinical evidence at my disposal. This evidence is of two types. For one thing, the notch repeatedly characterizes patients who in other respects appear free from cochlear damage. Second, Juers has pre-

sented data which suggest that bone conduction is improved in the fenestrated ear.[6]

My associates and I have analyzed Juers' data statistically. The pattern of improvement found in Juers' data is almost a direct reversal of the notch just mentioned. It is still too early to have a final opinion on this point, and research is continuing. However, as illustrated by figure 3, the clinical evidence is most provocative.

Figure 3 presents the preoperative and postoperative audiograms for an ear subjected to fenestration surgery.[7] One notes that both the air and bone conduction curves show postoperative improvement. The bottom chart in the figure compares the two bone levels. The shaded area shown in this chart represents my present estimate of the shift in

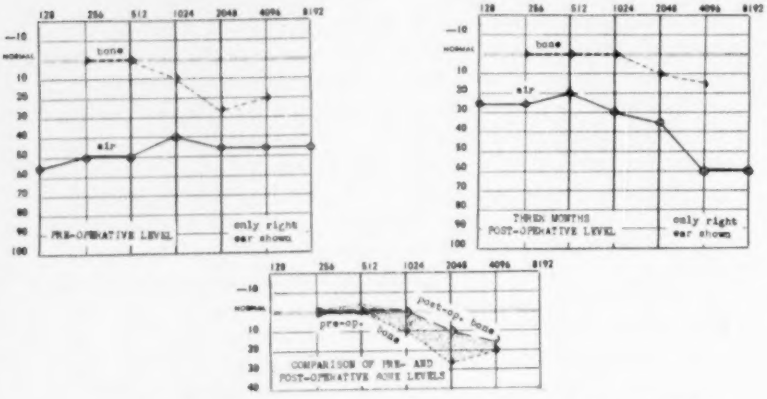


Fig. 3.—Audiogram illustrating shift in bone sensitivity after fenestration surgery; shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors; the postoperative bone curve shows improvement almost identical with the shaded area.

bone sensitivity owing to mechanical factors associated with stapedial fixation. One observes that the separation between the two bone curves corresponds very closely to the shaded area. This case offers one example of the clinical evidence which suggests the following conditions: (1) fixation of the stapes modifies the mechanical constants of the inner ear so that its frequency response to skull-borne vibration is changed; and (2) fenestration surgery restores the mechanical constants to approxi-

6. Juers, A.: Observations on Bone Conduction in Fenestration Cases, Ann. Otol., Rhin. & Laryng. **57**:28-40, 1948.

7. As with all cases reported here, tests were conducted under my supervision in the Hearing Clinic at Northwestern University.

mately their earlier values, so that there is a reversal in acuity for bone conduction stimuli.

The foregoing hypothesis does not invalidate the clinical premise that the bone conduction curve gives an indication of nerve function. One would expect changes in bone threshold due to mechanical factors to be minor in degree. These changes would be superimposed on loss for bone conduction due to inner ear impairment. For example, in cases in which stapes fixation is associated with inner ear deafness, the notch due to mechanical factors is added to the abnormality in the bone curve resulting from the perceptive lesion. This feature is illustrated by figure 4 *A*. The bone curve in this case may be considered a composite of a mechanically induced notch and an inner ear loss. The component representing the mechanically induced notch is indicated by the shaded area. The gap between the shaded area and the bone curve represents the component of inner ear deafness. Notice that when one corrects the air curve by an amount equal to this component, the resulting curve for the conductive component lies at a level and has a slope which is theoretically credible for a case of stapes fixation without cochlear damage. This case exemplifies superimposition of conduction and perceptive components in the probable presence of a small shift in bone sensitivity due to mechanical factors.

A more dramatic example is shown in figure 4 *B*. The diagnosis in this case was clinical otosclerosis with secondary nerve involvement. Notice the substantial parallelism in the troughs which appear on the air and bone curves. Again, the shaded area indicates the component of inner ear loss. This area may be subtracted from the bone conduction audiogram to yield the component of inner ear deafness. The conduction component may be considered as the difference between the air audiogram and the component of nerve loss.

Another interesting aspect of the audiometric contours characterizing otosclerosis involves the impedance formula discussed by Johansen [8] and others. I shall consider here only the shape of the air curve.

Figure 4 *C* presents the pure tone thresholds for a patient with clinical otosclerosis with unequal progress of the disease in the two ears. Notice that both ears show some notching of the bone curve. Notice further that the air conduction curve for the right ear rises for higher frequencies and reaches the normal line at 4096 cycles per second. In other words, this ear has been affected primarily in the low frequencies. According to Johansen's views, such a change should result from increased stiffness in the conduction mechanism. By contrast, the air conduction curve for the left ear is a composite of the configuration for

8. Johansen, H.: Relation of Audiograms to the Impedance Formula, Acta oto-laryng., 1948., supp. 74, pp. 65-75.

the right plus a depression of hearing for higher frequencies. The impedance formula accounts for an additional change of this type on the basis of a mass increase.

It would appear from such clinical evidence that the progression in stapes fixation occurring in otosclerosis is characterized first by increased stiffness and subsequently by increased mass. A sequence of this type is entirely logical. The initial stages of fixation do not fully preclude vibration but offer obstruction which would change the elasticity of the system. A stage must be reached, however, when fixation is extreme enough so

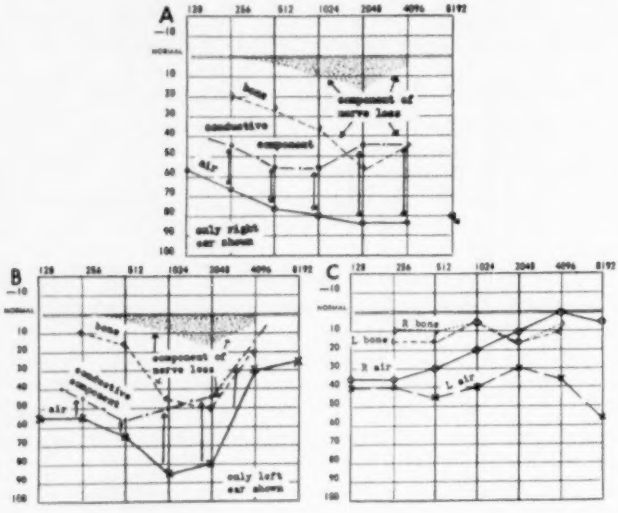


Fig. 4.—*A*, air and bone audiograms for right ear of a woman having clinical otosclerosis with secondary nerve involvement; conduction component is air curve minus component of nerve loss (i. e., gap between bone curve and shaded area); shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors. *B*, air and bone audiograms for left ear of a woman having clinical otosclerosis with secondary nerve involvement; conduction component is air curve minus component of nerve loss (i. e., gap between bone curve and shaded area); shaded area represents approximate depression in bone sensitivity to be expected from mechanical factors. *C*, air and bone audiograms for man with unequal progress of clinical otosclerosis in the two ears. The air curve for the right ear (partial fixation of stapes) reveals loss for low frequencies. This curve is suggestive of increased stiffness. The air curve for the left ear (stapes fixation) shows additional loss involving the high frequencies. The additional loss suggests increased mass, probably through loading the stapes with adjacent bone.

that (from the mechanical point of view) the stapes becomes loaded by the adjacent bony mass.

Proper audiometry can have great diagnostic importance when one is dealing with such a progression. It may encourage earlier and more definitive recognition of clinical otosclerosis. It may allow stages in the pathologic condition to be specified with greater surety. If the time ever comes when medical therapy for otosclerosis is developed, proper audiometry can encourage more effective therapy by aiding in early discovery of cases.

## CONCLUSION

The foregoing discussion has been presented partly for its illustrative value. We have only begun to take advantage of the contribution to diagnosis which bone conduction audiometry can make. The first step has been to perfect the methodology. Measurements which are clinically adequate can now be obtained if proper precautions are observed. However, both technical problems and a widespread mistrust must be overcome before bone conduction audiometry becomes widely useful. Furthermore, most of the finer diagnostic interpretations which air-bone relations may eventually allow still await discovery. Already some of these finer implications appear to have emerged in the study of clinical otosclerosis. Future research and clinical application will make bone conduction audiometry a diagnostic tool of increasing value to otology.

## DISCUSSION

### ON PAPERS BY DRS. SILVERMAN AND CARHART

Dr. Hallowell Davis, St. Louis: Last summer Dr. Juers sent me some of his measurements of bone conduction threshold before and after fenestration. I was skeptical that an improvement occurred as the result of operation, although my own analysis of his data indicated a statistically reliable effect at 2000 cycles per second. I expressed my skepticism to Dr. Juers, but now, with the additional data and brilliant analysis presented by Dr. Carhart, my doubts are set at rest. I believe Dr. Carhart has made an important contribution to the interpretation of bone-conduction audiograms and has explained one of the important causes of the familiar irregularity and implausibility of such data.

It was because of such irregularities and uncertainties that we at the Central Institute for the Deaf and the McMillan Hospital have tried hard to avoid the use of bone conduction measurements in evaluating the success of the fenestration operation and in making prognoses. We have, however, been unable to find a better substitute, and, in a paper just completed for *Laryngoscope*, Dr. Walsh and I have used preoperative bone conduction data. We recognized implicitly that the effect described by Dr. Carhart might occur and allowed for it by taking as our "normal" preoperative values the average for patients who were proved by the subsequent success of the fenestration operation to have normal cochlear function. We failed to find, however, the improvement in bone conduction threshold described by Dr. Carhart, because we did not determine bone conduction thresholds after operation.

Our analysis of the improvement in hearing after fenestration is pertinent to Dr. Carhart's observations and also illustrates several of the principles reviewed by Dr. Silverman. I shall, therefore, summarize the highlights of the paper by

Dr. Walsh and me (Davis, H., and Walsh, T. E.: The Limits of Improvement of Hearing Following the Fenestration Operation, *Laryngoscope* **60**:273-295, 1950).

The hearing losses for speech have been measured before and shortly after (about three months) 195 consecutive fenestration operations.

The data are compared with the results of an analysis of known physical and psychoacoustic factors involved. They are in excellent agreement and support the following propositions.

1. The best result to be expected after fenestration is a residual hearing loss for speech of 20 decibels. This residual loss depends primarily on absence of the ossicles.

2. The residual hearing losses of cases of successful fenestration scatter at random about a mean. The scattering is due to following reasons: (*a*) the "error of measurement" of the hearing test; (*b*) normal variations in the acuity of the inner ear, and (*c*) variations in the physical properties of the patients' skin flaps and other tissues and uncontrolled minor variations in surgical procedure.

3. The maximum possible conduction hearing loss for speech is about 60 decibels. When speech is more than 60 decibels above normal threshold it can be heard by bone conduction. Losses greater than 60 decibels therefore mean that more or less nerve deafness must be present. This is confirmed by bone conduction audiometry.

4. Nerve deafness is not alleviated by the fenestration operation. The maximum gain that can be expected from fenestration is therefore 60 decibels minus 20 decibels, i. e., 40 decibels.

5. The residual hearing to be expected after fenestration depends on the amount and character of nerve impairment (if any) but not on the amount of preoperative conduction deafness.

The number of decibels by which a patient's immediate postoperative hearing loss for speech exceeds the unavoidable minimum we call the "surgical deficit." For pure conduction deafness the deficit is the excess above 20 decibel residual loss. For mixed deafness additional allowance is made for the "nerve" component of the deafness. The latter is at present best measured as the hearing loss by bone conduction. Several difficulties attending such measurements are discussed.

The mean postoperative hearing loss for our 69 patients with pure conduction deafness operated on since Dec. 1, 1947, excluding definitely unsatisfactory cases with final losses greater than 35 decibels, is 26.4 to 3.3 decibels.

The incidence of failure, the mean surgical deficit for the remaining cases, and its standard deviation are three appropriate measures (among others) for evaluating a surgeon's technical proficiency. If accurate estimates of nerve impairment are available one can, from these measures of the surgeon's skill, make prognoses of known probability. Thirty (15.7 per cent) of the patients of our series had an immediate postoperative loss for speech of more than 40 decibels. Fourteen of these "failures" are due largely or wholly to the presence of significant nerve impairment.

Failure of the operation in 12 patients (6.3 per cent of the series) is due to known or probable surgical "accidents," usually bleeding into the fenestra, as from otosclerotic bone. Failure in only 3 (1.6 per cent of the series) is completely without explanation.

# MASKING AND SHADOW HEARING IN BONE CONDUCTION

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AND
MATTHEW S. ERSNER, M.D.
PHILADELPHIA

IN THE performance of a hearing test the object of masking is to keep one ear occupied while the other ear is being tested. A complex noise consisting of the essential frequencies gives a satisfactory masking effect. The specific areas on the basilar membrane will resonate to the complex masking sound and will become refractory to stimulation from vibrations applied to the other ear.

If one ear is totally deaf and the other ear has normal hearing, a sound of adequate intensity applied to the deaf ear will register an auditory sensation. It is shadow hearing, the sound having traversed across the skull to stimulate the functioning basilar membrane. What is the route of that sound? Through the air receiver, it begins at the external acoustic meatus where the magneto-dynamic receiver is applied. The direct path leads the sound waves into the middle ear and from there into the insensitive inner ear, which is located in the petrous pyramid. Intervening between the two inner ears is the basilar part of the occiput, which is 2.3 cm. wide at this point. Through such a narrow bar of solid bone, stimulation of the functioning ear on the opposite side will be readily facilitated. By bone conduction, the path of the sound vibrations begins at the mastoid process and proceeds through the trabeculae in the subaditus region [1] or through the semicircular canals [2] to initiate the compression wave in the scala vestibuli. In the case of shadow hearing, when the bone conduction is applied to the deaf ear, it is logical to assume that the sound vibrations exert their effect on the mastoid of the opposite side by way of the squama occipitalis at the level of the inferior nuchal line, which is the shortest distance between the two mastoids. It measures 16.3 cm. It must be inferred, therefore, that the mechanism of shadow hearing by bone conduction is quite different from that of air conduction.

In clinical practice, we observed that masking is not completely effective in eliminating shadow hearing by bone conduction. On the other

From the Department of Otorhinology, Temple University School of Medicine.

1. Guild, S. R.: Ann. Otol., Rhin. & Laryng. **45**:736, 1936.
2. von Békésy, G.: Ann. d. Phys. **13**:111, 1932.

hand, in most cases, the ablation of shadow hearing through an air receiver is accomplished quite readily by masking. Figure 1 presents the masked bone conduction curves of a representative patient who was shown to have total unilateral deafness by air conduction, on the application of a masking sound to the healthy ear. We studied 3 other patients with unilateral deafness whose audiometric patterns presented practically identical features. To begin with, each one had normal air and bone acuity in his good ear. In each instance, an auditory sensation of an elevated threshold was registered when the bone receiver was applied to the deaf ear, while a complex masking sound was placed at the external auditory meatus of the hearing ear.

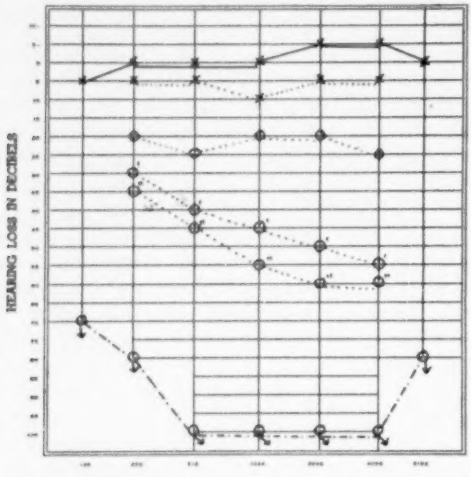


Fig. 1.—Audiogram showing unilateral deafness. The circle indicates the right ear; the cross, the left. The solid line indicates air and the dotted line bone conduction. The line of circles, dashes and dots indicates air conduction in the right ear with masking of the left ear by complex noise 75 decibels in intensity; the line of single accented circles and dots indicates bone conduction in the right with masking of the left ear by complex noise 75 decibels in intensity; the line of double accented circles and dots indicates bone conduction in the right ear with masking of the left ear by complex noise 100 decibels in intensity. This 14 year old patient had a total loss of hearing in the right ear since the age of 3 following an obscure febrile attack. Otoscopy and nasopharyngoscopy disclosed no abnormality.

In practice, we encountered an occasional patient with unilateral deafness who, in spite of masking, manifested a slight degree of shadow hearing even by air conduction. It may be attributed to the psychologic factor of selective hearing. As only the lower tones at the highest intensities are heard, this finding is of little practical significance in the appraisal of a clinical case.

Experimentally, we tried to bring about the ablation of auditory sensation by bone conduction through the application of a complex masking sound to each external auditory meatus. Our subjects were 10 young adults—5 male and 5 female—who had no history of aural disease and whose hearing was normal. In these experiments, bone receiver 726 A with Western Electric 6 B.P. audiometer was used, and for masking we constructed a double receiver for the Pilling-Witting masker. In none of our 10 subjects were we able to bring about the ablation of auditory sensation by bone conduction, even if the bilateral masking was raised to the intensity of 100 decibels. There was, however, a threshold elevation with the increase in the loudness of the masking sound. Figure 2 shows the mean bone conduction curves in the presence of bilateral masking with intensities of 75 and 100 decibels.

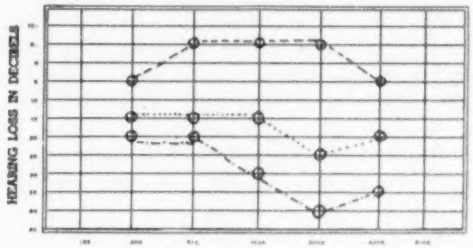


Fig. 2.—Audiogram showing the effect of bilateral masking on acuity of hearing by bone conduction. The broken line indicates the normal bone threshold; the dotted line indicates bone threshold when a complex noise of 75 decibels is applied to each external auditory meatus; the line of dashes and dots indicates bone threshold with bilateral masking of complex noise of 100 decibels.

## COMMENT

A vibrating body applied to any part of the skull must necessarily cause a widespread disturbance. If the force of the vibrations is great enough, even remote and partially refractory receptors will be stimulated. When, because of activation by other sound waves, some nerve fibers in one or both ears become totally refractory, other nerve fibers take on the oncoming sensation. The modern physiologic concept is that nerve fibers "alternate and rotate" in performing their function. To bring about the total ablation of sensation in a peripheral nerve, the stimulus must be so massive as to cause absolute refractoriness in every available nerve fiber. In the case of the ear, a stimulus of such great intensity will exceed the threshold of pain.

The concept of "point of maximum vibration," referable to each resonator on the basilar membrane, has been formulated by Gray.[3] His modification of the Helmholtz theory implies that those fibers of the basilar membrane which are in tune with the presenting sound are set into maximum vibration, and that for a variable distance on either side of the resonator the nerve fibers vibrate with a decreased intensity. In the light of this theory, the type of refractoriness, to which the basilar membrane is subject, depends largely on resonance. A tone of minimum audibility causes absolute refractoriness in the nerve fiber which vibrates in tune with it, while a sound wave of an intensity above threshold will bring about absolute refractoriness in its resonator and relative or partial refractoriness in the nerve fibers lying on either side of the resonator.

The object of masking is to create refractoriness to shadow hearing. This can be done either by the sheer force of a massive stimulus or through the effect of resonance, which is the basis of the modern clinical procedure. Shadow hearing will not be heard by the basilar fiber which vibrates at its maximum in response to a masking tone, and only sounds louder than threshold will register an auditory sensation in the fibers lying on either side of the resonator. Absolute refractoriness depends on absolute resonance, and the farther apart in frequency the shadow tone is from the masking sound, the more discernible will it be.

As to the extent of ablation of shadow hearing by a complex sound of 70 to 75 decibels applied to the hearing ear, several factors must be considered. A shadow tone of a different musical quality than the masking sound will be ablated to a lesser degree by virtue of the psychologic attribute of selective hearing. The mechanism of transmission of a shadow tone influences its audibility. It has been shown by Stevens[4] that pitch increases with intensity for high tones and decreases with intensity for low tones. In other words, sound pressure modifies the frequency of a tone. At its propagation and at its destination, the same tone may show a variation in frequency, depending on the differential in sound pressure, whether, in transit, it gained or lost in intensity. A fixed frequency applied to the deaf ear will undergo a variation, no matter how slight, by passing through the skull. At its destination—the hearing ear—it will not meet absolute refractoriness, owing to the masking sound, but relative or partial refractoriness; and if loud enough, the shadow tone must register an auditory sensation. It is also obvious that the changes in frequency and quality the shadow tone will undergo will vary, whether it travels in a direct path from a magneto-dynamic

3. Gray, A. A.: J. Anat. Physiol. **34**:324, 1900.

4. Stevens, S. S.: J. Acoust. Soc. Am. **6**:150, 1935.

receiver into the middle ear containing ossicles with perfect leverage and from there into the base of the skull, where a 2.3 cm. solid bar of bone separates the two inner ears, or whether it travels in an indirect path, an oscillator setting the bones of the skull into vibration and initiating a compression wave by way of the mastoid of the opposite ear.

Elevation of threshold, due to masking, is essentially a feature of partial or relative refractoriness. In experimental psychology, under ideal laboratory conditions, absolute refractoriness may be demonstrated. In clinical practice, with the available paraphernalia, the elevation of the threshold, obtained through masking, is indispensable for diagnostic purposes. The masked air conduction audiogram has a greater elevation of threshold than the masked bone conduction curve. As the effect of masking on bone conduction is rather variable, the results of this procedure are most useful when evaluated in conjunction with other clinical criteria, notably the masked air conduction threshold.

1923 Spruce Street.

1915 Spruce Street.

# HOARSENESS IN CHILDREN

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NEW YORK

HOARSENESS in children is not a new subject, although it has been sadly neglected by most laryngologists. Thanks to the efforts of Dr. Francis W. White,[1] who has been greatly interested in this problem, attention has again been directed recently to young children presenting hoarseness.

The importance of this problem has been recognized also by Dr. Letitia Raubicheck, director of speech improvement, in the department of education of New York city. With intelligence and aggressiveness, she has approached this problem in a manner which speaks well for the future care of such young, afflicted patients. We, at our special laryngeal and speech department at St. Vincent's Hospital, are indeed grateful to her for the privilege of seeing and studying young subjects with this condition.

Hoarseness in children, while widespread, does not present the bizarre and extensive etiologic factors noted in hoarseness in adults. Neither neurologic nor pulmonary pathologic conditions, such as new growths, cysts or aneurysms which produce paralysis of a vocal cord, are frequent causative factors.

The method of study we use is not complicated, but it is predicated on sound, practical principles. First, a complete history is obtained on a special chart. Then a complete medical examination is made, with particular reference, of course, to the head and neck. Patients requiring dental, neurologic or pediatric consultations are referred to the specific department.

After this examination, or medical screening, the patient is classified on the basis of whether his problem is purely laryngeal or one requiring speech therapy.

Laryngeal examination in the child is not as difficult as one would suppose. In fact, we find it much easier in many instances than examination of the adult larynx. It should be borne in mind that the child's larynx is situated higher in the neck than it is later in life and that this

From the Department of Otolaryngology, St. Vincent's Hospital.

Presented before the Section on Otolaryngology, New York Academy of Medicine, Oct. 20, 1948.

1. White, F. W.: Some Causes of Hoarseness in Children, Ann. Otol., Rhin. & Laryng. **55**:537-542 (Sept.) 1946.

situation is conducive to quick visualization of the vocal cords. The pathologic change of the vocal cord is then quite easily recognized.

As with adults, we have two methods of examination at our disposal, the direct and the indirect. In the indirect examination the method of

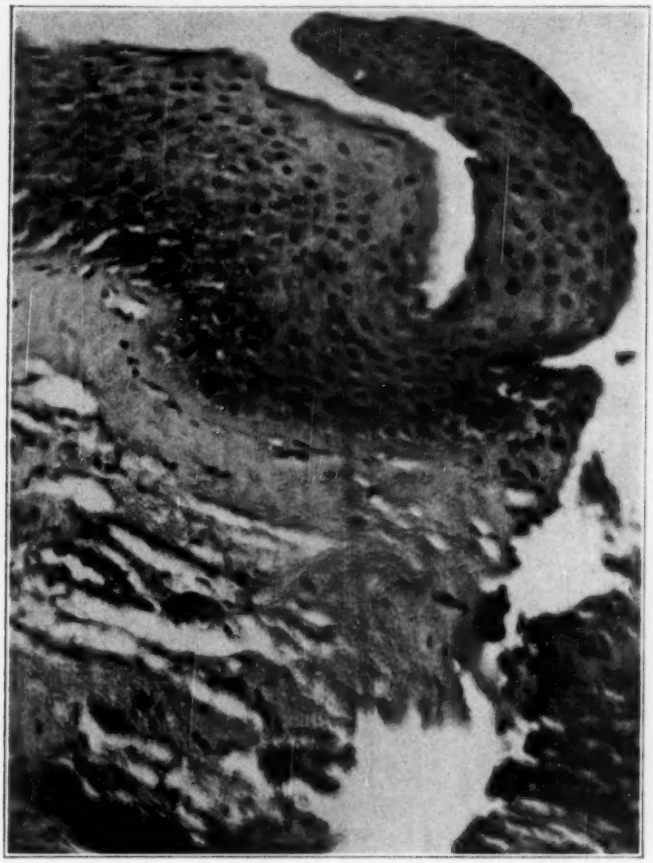

Fig. 1.—Section through a small polypoid nodule. The surface is lined with thick stratified squamous epithelium, which has penetrating pegs in places. The stroma is boggy and, in general, quite loose, presenting the picture of polyposis.

approach is of great importance. Kindness and gentleness are the key words to this approach. We try to gain the confidence of these little patients. Most of them are old enough to understand what you intend

to do, and if time is taken to explain to them what you propose to do, considerable cooperation will be forthcoming. In many instances an

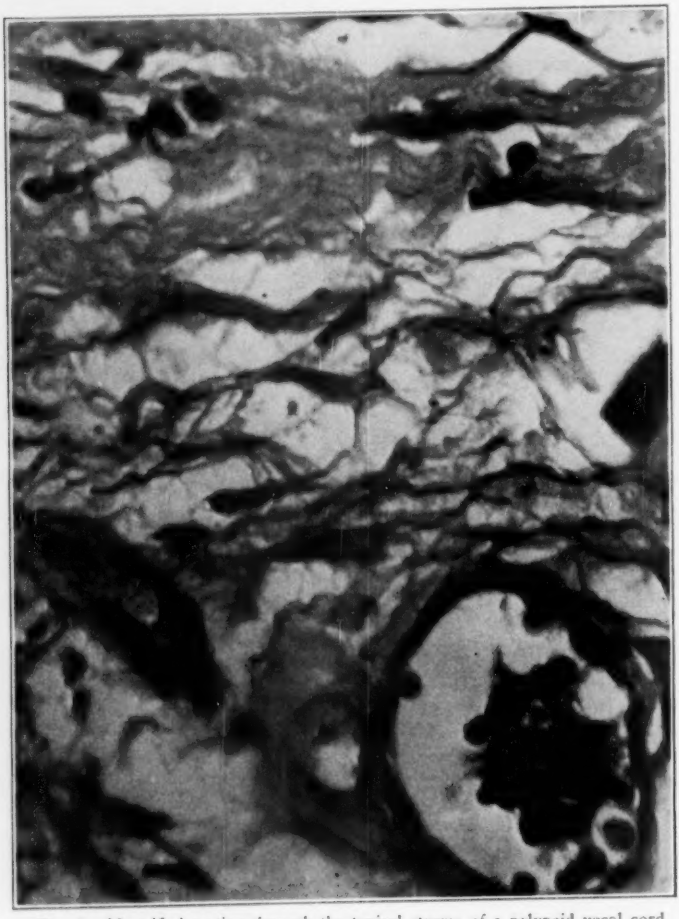

Fig. 2.—Magnified section through the typical stroma of a polypoid vocal cord. It is edematous; the collagen fibrillae are frayed; the blood vessels are increased in number and small, some of them showing a deposition of hyalin below the endothelium, with narrowing of the lumen. There is an occasional infiltration with round cells. The mucin has degenerated to form little cystic cavities. Fibrinoid degeneration is present with autolysis of the connective tissue.

initial swabbing of the soft palate and pharyngeal wall with a weak anesthetic, supplemented by a light application of this same agent to

the under surface of the tip of the tongue, helps tremendously. Naturally, a small laryngeal mirror or pharyngeal mirror is used. Speed, precision and gentleness are requisites.

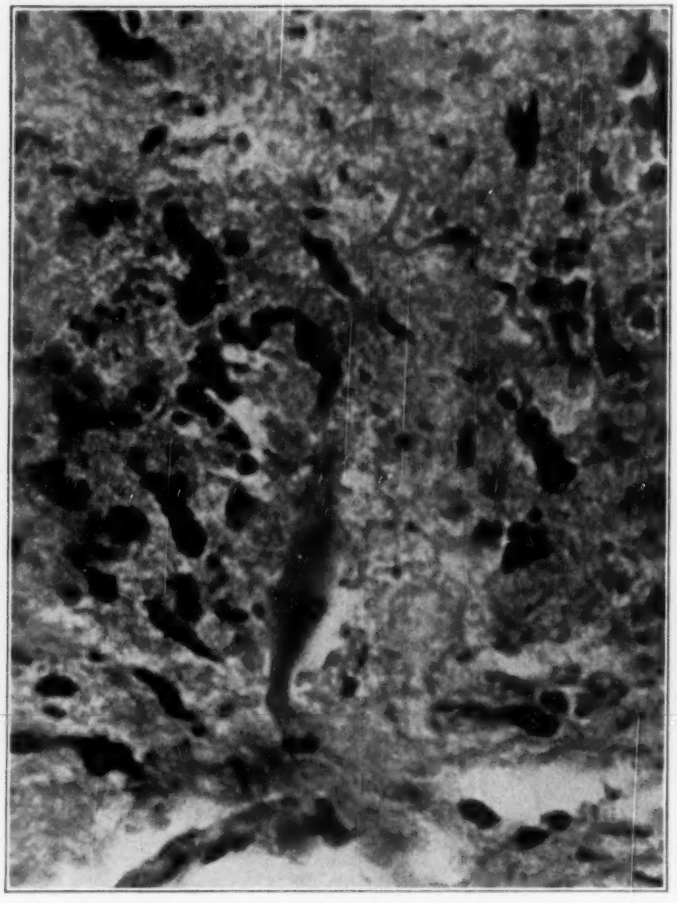

Fig. 3.—Section through stroma of a typical papilloma. In contrast to the stroma of polyposis, the stroma of papilloma is considerably denser, with little to no edema. Round cells can be noted.

However, there are patients for whom examination by the indirect method is not successful. In such cases we recommend a direct laryngoscopy, with general anesthesia and with the definite understanding

that if we find a laryngeal pathologic condition amenable to operation, we will proceed to operate. Occasionally, a hoarse patient needs to have the tonsils and adenoids removed. In such cases the larynx is

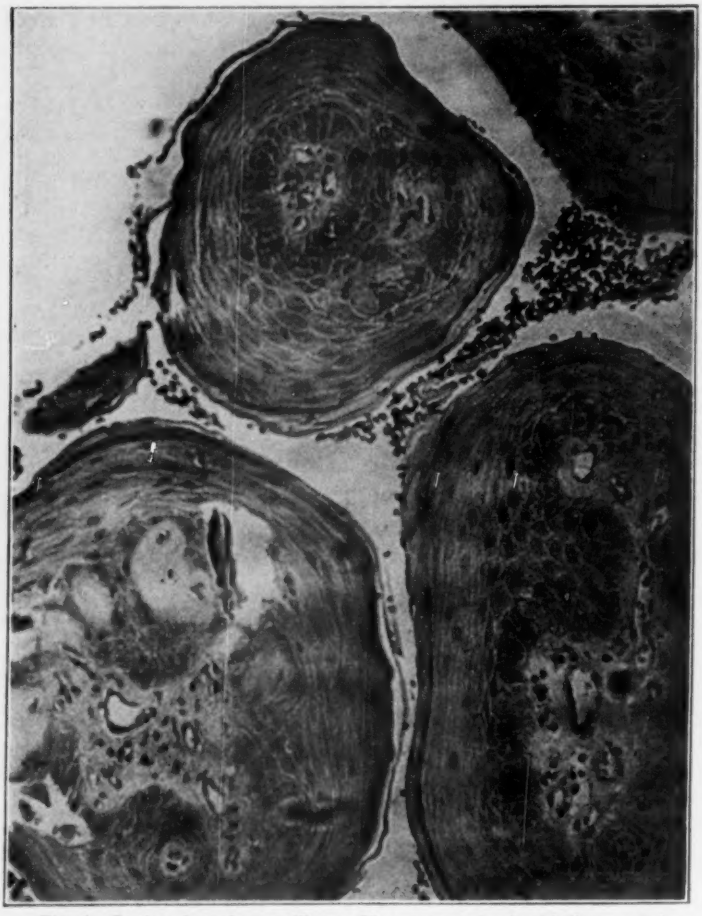

Fig. 4.—Section through a papilloma. The overgrowth of epithelium as it is thrown up into many folds is well demonstrated. The epithelium is thick with a deposit of keratin on its surface.

examined at the time of the operation. We prefer to perform this examination at the conclusion of the removal of the tonsils and adenoids, although we can see no objection to doing it first. As a preliminary

to any procedure, we make voice recordings and repeat them at various intervals after treatment or operation. Thus we have an audible record of the progress in each case. Drawings are made of the findings at examination or operation and a careful follow-up system is employed.

Fig. 5.—Section through an epithelial layer with a thick coating of keratin. The papillomas have tremendous thickening of the epithelial layer, an example of parakeratosis.

In addition to the young subjects referred by Dr. Raubicheck, the other patients are from the regular ear, nose and throat clinic, the

pediatric clinic and the neurologic clinic at St. Vincent's Hospital. All patients with chronic hoarseness are thus sent to the laryngeal clinic.

This study is concerned chiefly with those patients up to and including the age of 14 who have been examined in this special laryngeal department. This group does not include patients with hoarseness due to acute laryngeal lesions. Hence patients with acute laryngotracheobronchitis and acute laryngitis have been excluded.

This report—which is necessarily preliminary—concerns 56 patients. There is no special basis for selection except that previously mentioned.

TABLE 1.—*Clinical Classification of Causes of Speech Disorders of 56 Children Aged 14 and Younger*

| Laryngeal Pathologic Conditions | Malocclusion | Tongue Tie | Cleft Palate | Postinfectious Lesions of the Central Nervous System | Social Problems | Mental Deficiency | Stuttering |
|---|---|---|---|---|---|---|---|
| 19 | 8 (2 with l.p.c.*) | 4 | 2 | 2 | 3 | 6 (1 with l.p.c.*) | 16 (3 with l.p.c.*) |
| | | Deaf Mutism | Impaired Hearing | | Unclassified | | |
| | | 1 (1 with l.p.c.*) | 1 | | 1 | | |

* l.p.c. indicates laryngeal pathologic conditions.

TABLE 2.—*Clinical Classification of 19 Patients with Laryngeal Pathologic Conditions*

| Lesions of the Vocal Cord | Thickening of Cord with Polyposis | Papillomas | Small Band at Anterior Commissure |
|---|---|---|---|
| 17 | 14 (6, bilateral); (1, operation performed) | 2 (bilateral operation performed) | 1 |
| Edematous Ventricular Bands | | Loose Corniculate and Cuneiform Cartilages | |
| 1 | | 1 | |

Table 1 shows the etiologic categories of the voice or speech defects of the group. Nineteen of these patients had laryngeal pathologic conditions. The causes of the speech defects of the other patients were malocclusion, tongue tie, cleft palate, postinfectious lesions of the central nervous system, social problems, mental deficiency, pure stuttering, deaf-mutism and impaired hearing. The latter group was treated by the dentists, neurologists, pediatricians and speech therapist at our hospital.

Table 2 is a clinical classification of the 19 patients with laryngeal pathologic conditions.

Seventeen patients had lesions of the true vocal cords; the cords of 14 of these revealed thickening and polypoid changes, which were bilateral in 6. Two patients had multiple papilloma, bilateral in both;

1, a small band at the anterior commissure; 1, edematous ventricular bands, which closed on phonation before the true vocal cords did, and 1, loose corniculate and cuneiform cartilages, which had a tendency to be drawn into the larynx on inspiration.

Seven of the 19 patients with laryngeal pathologic changes had speech defects from other causes as well, malocclusion, deaf-mutism, stuttering and mental deficiency.

This study is chiefly concerned with those patients manifesting pathologic changes of the true vocal cords, the object being that such pathologic conditions must be eradicated before functional speech therapy is started. This stipulation is of paramount importance. With a pathologic condition of the larynx no progress can be achieved until that condition is eliminated.

## METHOD OF TREATMENT

If the thickening of the vocal cord was minimal, conservative treatment was instituted. This treatment consisted in functional rest, use of *p*-chlorophenol spray and for the sinus, nasal tamponade, postnasal irrigations and use of sulfathiazole sprays and vasoconstrictors. Only 1 patient of the 19 with laryngeal pathologic changes could actually be classed as cured as a result of this regimen; several others showed some improvement, and the remainder were advised to undergo surgical procedure. Only 6 submitted; of these, 2 had multiple papilloma and 4 had polyposis.

In the laryngeal operation the Loré[2] stripping technic was used. A mixture of ether and oxygen was used to induce anesthesia. With the Imperatori subglottic forceps, the growth on the vocal cord was removed in toto through an anterior commissure speculum. Only one cord was stripped at a time to prevent web formation. If the lesion is bilateral the other cord is stripped about a month later. The patient is hospitalized only overnight and is allowed to use his voice immediately after the operation. In every case definite improvement was reported, sometimes as soon as the day after operation, sometimes as long as two weeks later. Maximum improvement was attained in about one month. In the more recent cases, preoperative recordings were made of the patient's voice to demonstrate the improvement. Recordings, incidentally, are utilized in the routine of young patients with speech defects of other causes as well and in the preoperative routine of our adult patients.

Microscopic examination of the serially sectioned specimens, which were reviewed by Dr. Rottino, of the pathology department, revealed two predominant pictures: polyposis and papillomas.

The slide of a typical polypus showed a nodule, the surface of which was lined with thick stratified squamous epithelium. In some nodules

2. Loré, J. M.: Stripping of the Vocal Cords, Arch. Otolaryng. **42**:378-383 (Nov.-Dec.) 1945.

the epithelium was proliferating and forming deeply penetrating pegs. The stroma was edematous, appearing water-logged; the collagen fibrillae were frayed. The blood vessels were increased in number and were small; some of them showed a deposition of hyalin below the endothelium and narrowing of the lumen. Edema was present beneath the endothelium of some blood vessels. The stroma, in addition, showed an occasional infiltration with round cells, whereas in some specimens the mucin had degenerated to form little cystic cavities. Fibrinoid degeneration was present.

The sections of the papillomas showed a pronounced overgrowth of epithelium, which was thrown into many folds. On the surface epithelium was a deposit of keratin, presenting the picture of parakeratosis. The stroma was more compact, less edematous, if edematous at all, in comparison to that of the polypi.

In general, these changes, particularly those seen in the polypi, are not unique to the vocal cords. Similar changes are seen on diseased heart valves, in cystic degeneration of the uterus and even on dissecting aneurysms of the aorta. Chronic irritation was apparently the cause in the pathogenesis.

After operation, the patient, in addition to being followed by the laryngologist, was treated by the speech therapist to insure proper use of the voice. The results have been gratifying.

One of the tantalizing aspects surrounding this problem of hoarseness in children is the etiologic question. Nineteen subjects are actually an insufficient number for postulating any definite conclusions. However, as an indication of predisposing causes, about 37 per cent had on examination, or had had on history, a severe infection of the respiratory tract; 42 per cent, enlarged tonsils and adenoids, and 24 per cent, deviation of the nasal septum. These percentages do not total one hundred, since many had a combination of these conditions, which were about equally divided among the male and female patients. There was a definite familial history of hoarseness in 15 per cent. Another possibly interesting finding was that 52 per cent of these patients were of Latin parentage, and the Latin people have a known habit of overuse or abuse of the voice.

In other words, almost all of this group had either a history of infection of the respiratory tract, severe or frequent, or lived in an environment conducive to overuse of the voice. Could these be the predisposing or etiologic agents which are responsible for the polypi or papillomas on the true vocal cords? When more cases are assembled, the answer may be clearer. In the interim, as more children with hoarseness are given an adequate medical examination, which includes a competent laryngologic examination, the pathologic condition will be recognized and then treated in an intelligent manner.

REPORT OF SIX CASES REQUIRING LARYNGEAL SURGERY

M. C., a white girl aged 7½, was first seen in the laryngeal clinic in July 1947, with the chief complaints of hoarseness and inaccurate speech since she started to talk. Two siblings had normal voice and speech. The patient was in a class at school for the mentally retarded and also attended a speech class once a week. Her history indicated frequent sore throat and colds, and her mother stated that the patient screamed a great deal.

On physical examination, enlarged tonsils and adenoids and carious teeth were seen, and on indirect laryngoscopic examination, a nodule was seen on the right vocal cord. Speech treatment was started at the hospital clinic, with little improvement, and in November tonsillectomy and adenoidectomy were performed. The patient continued to attend the hospital speech clinic. On repeated indirect laryngoscopic examinations, the nodule of the right vocal cord was seen to persist and, finally, to become more prominent.

In April 1948, there was an additional slight thickening of both vocal cords at the middle third. In May, the right vocal cord was stripped, with the patient under general anesthesia. The clinical and pathologic microscopic diagnosis was polyposis. Two months later the right vocal cord was found to be well healed and the patient's voice slightly improved. However, the thickening of the left vocal cord became more prominent, and here too a nodule was found.

In August, stripping of the left vocal cord was done with the patient under general anesthesia. At this time a preoperative recording of the voice was made. Clinical and pathologic microscopic diagnosis again was polyposis. Several days after operation, the mother of the patient stated that the child's voice had definitely improved. The patient was last seen in November 1948; her voice was very good, the edge of right vocal cord was straight and the cord was of normal thickness and was slightly injected. The left vocal cord showed a slight convexity in the middle third and slight thickening.

F. S., a white boy aged 10, was first seen in the laryngeal clinic in June 1948, with the chief complaint of hoarseness of one month's duration which had progressively become severer. The patient had had a tonsillectomy and adenoidectomy at the age of 3. His mother stated that he shouted a great deal. Physical examination indicated nothing essentially abnormal, but indirect laryngoscopy revealed a small polypoid growth at the anterior third of the right vocal cord. Ten days later the patient was admitted to the hospital and, under general anesthesia, stripping of the right vocal cord was performed. The pathologic microscopic diagnosis was polyposis. One month after the operation the mother stated that the patient's voice had definitely improved. The right vocal cord was straight, but there was some thickening of the left vocal cord. The patient was last seen in March 1949. Indirect laryngoscopic examination revealed slight thickening of the right vocal cord, while the left cord appeared normal; the thickening that had been previously noted had regressed. The mother stated that she was very pleased with the patient's voice.

S. B., a white girl aged 11, was first seen in the laryngeal clinic in March 1948, with a history of hoarseness and some difficulty in breathing since the time that she first attempted to speak. The patient has been a deaf-mute since birth. The mother stated that the child screamed a great deal during babyhood and had had repeated infections of the upper respiratory tract. Physical examination showed normal drum membranes. Indirect laryngoscopic examination revealed that both vocal cords were papillomatous, the left cord presenting a large, somewhat pedunculated mass at its posterior third. The patient had the typical speech of the deaf. The results of the remainder of the physical examination were essentially normal. One month later, the left vocal cord was stripped with the patient under general anesthesia. The pathologic microscopic diagnosis was papilloma.

In July there was noted a small tab of tissue on the right vocal cord subglottically at the junction of the anterior and the middle third. The left vocal cord was healing satisfactorily. At this time the patient was readmitted to the hospital, a voice recording was made and, under general anesthesia, the right vocal cord was stripped and the subglottic tab of tissue removed. The pathologic diagnosis was polyposis. Two and a half months later, the parents stated that the patient's voice was definitely not as husky as before and that she breathed more easily. Indirect laryngoscopic examination revealed that both vocal cords were dull red, somewhat thickened and appeared shortened. The patient was last seen in January 1949, and the clinical and laryngeal pictures were satisfactory.

R. P., a white girl aged 14 was first seen in the laryngeal clinic in May 1948, with the chief complaints of hoarseness and stuttering since early childhood. The patient has been a mild stutterer since the age of 8. Physical examination indicated nothing essentially abnormal except carious lower molars. On indirect laryngoscopic examination, a small growth was found at the middle third of the right vocal cord. The remainder of that cord was polypoid: the left vocal cord was normal. The isthmus of the thyroid was easily palpable. In July, she was admitted to the hospital and, under general anesthesia, the right vocal cord was stripped. Pathologic diagnosis was polyposis.

A recording of the voice had been made preoperatively. Postoperatively, the patient attended the hospital speech clinic, where an attempt was made to teach her the use of the extrinsic muscles of the larynx. Three months postoperatively, an indirect laryngoscopic examination showed a slight irregularity of the right vocal cord. However, on phonation, the vocal cords approximated well. The patient stated that she noted improvement in her voice and speech; her teacher at school had also told her that her voice had improved. She was last seen in November 1948, at which time her voice was decidedly improved, even though she had an infection of the upper respiratory tract. The right vocal cord was straight and slightly injected.

W. E., a Negro boy aged 12, was first seen in the laryngeal clinic in May 1948, with a history of hoarseness of about one year's duration. His mother stated, however, that he probably had had a husky voice for several years but that she had not noticed it particularly. Indirect laryngoscopic examination revealed thickening of the anterior third of both vocal cords, with more marked thickening of the right. The results of the remainder of the physical examination were essentially normal. The patient was treated conservatively with *p*-chlorophenol nose and throat spray. By July there was still no change in the laryngeal picture. One month later the patient was admitted to the hospital. A voice recording was made, and the patient was then taken to the operating room, where, under general anesthesia, the right vocal cord was stripped. Pathologic microscopic diagnosis was polyposis.

The postoperative examination showed that the edge of the right vocal cord appeared smooth and somewhat edematous. There was little improvement in the voice. The patient was then readmitted to the hospital in December. On indirect laryngoscopic examination the left vocal cord appeared polypoid. The tonsils were hypertrophied. He was taken to the operating room again, and, under general anesthesia, a tonsillectomy and a stripping of the left vocal cord were performed. The pathologic microscopic diagnosis of the specimen removed from the left vocal cord was polyposis. Four days postoperatively, the patient was seen in the clinic and a remarkable improvement in the voice was evident. He was last seen in January 1949, at which time he had an infection of the upper respiratory tract. His voice was improved, but there was some slight irregularity of the left vocal cord.

C. H., a white boy aged 4, was first seen in the laryngeal clinic in July 1946, with the chief complaint of an exceptionally hoarse voice since birth. Lately, there had been accompanying difficulty in respiration.

The past history indicated that the child had been a premature baby and when first seen in the pediatric clinic at the age of 2 months was noted to be very small and undernourished. He had had more than the usual bouts of infections of the respiratory tract, and, at the age of 2, had been hospitalized for acute suppurative otitis media, acute nasopharyngitis and acute tonsillitis.

When admitted to the hospital from the laryngeal clinic in July, the patient was taken to the operating room; general anesthesia was supplemented with 4 per cent cocaine solution, which was swabbed in the larynx. Direct laryngoscopy showed multiple papillomas on both vocal cords and the left ventricular band. There was pronounced obstruction to the laryngeal airway. With the Loré technic, both ventricular bands and both vocal cords were stripped. The anterior end of the left vocal cord was not operated on. Because of the extensive surgical procedure, the patient was kept in the hospital for repeated direct laryngoscopies and repeated dilation of the larynx to prevent web formation. One month later the patient was discharged, at which time a no. 24 laryngeal sound was easily passed and the vocal cords appeared to be in excellent condition. In April 1947, the patient was again admitted to the hospital with the complaints of difficulty in breathing and hoarseness. He was immediately taken to the operating room and, under general anesthesia, a direct laryngoscopy was again performed. A small loose growth was noted on the anterior third of the left vocal cord and was removed by means of a subglottic forceps. Pathologic microscopic diagnosis was papilloma. Eight months later the patient was readmitted to the hospital with the complaint of several attacks of tonsillitis associated with infections of the upper respiratory tract. A tonsillectomy and adenoidectomy were performed.

The patient was readmitted to the hospital in April 1948, again with the chief complaint of hoarseness. Direct laryngoscopy revealed that both vocal cords were slightly thickened, although no definite papillomas were seen. However, some tissue was removed from the left vocal cord. Two months later clinical evaluation showed that the patient's voice was better. He was then sent to the speech therapist, and his speech gradually improved. Numerous repeated clinical examinations of the nose, throat and larynx revealed infections of the upper respiratory tract with sinusitis. The mother stated that his voice was definitely clearer than before operation but that it lapsed into bouts of hoarseness with each flare-up of an infection of the upper respiratory tract.

In March 1949, because of repeated attacks of sinusitis, the patient was placed on treatment with nasal suction. At this time, although the arytenoid cartilages were injected, the vocal cords themselves appeared straight. Continued examination of the larynx by the indirect method until the last examination in June 1949 showed no evidence of any recurrences of any type of growth on the vocal cords or ventricular bands. The voice was clear, and on phonation there was good approximation of the vocal cords.

## CONCLUSION

Hoarseness in children is far more widespread than is usually supposed. The larynxes of such patients very often present definite pathologic conditions which are amenable to operation.

All children with hoarseness should therefore have a competent and complete laryngeal and medical examination.

325 West End Avenue.

# STREPTOMYCIN IN TREATMENT OF TUBERCULOUS OTITIS MEDIA

## An Evaluation

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DURING the past decade a new type of weapon has appeared to be used in the battle against various micro-organisms which have invaded the human body. This weapon is the antibiotics. As each antibiotic has appeared, it has been hailed as the medical version of the philosopher's stone, but closer investigation revealed its shortcomings. One of these greeted with much enthusiasm at first is streptomycin, and it was believed that a real conqueror of the tubercle bacillus had been found. Now that this substance has been used for a sufficient time, one should be able to evaluate its efficacy.

Judging from articles and texts before the advent of streptomycin, the prognosis for a patient with tuberculous otitis was not favorable. Phillips [1] stated that "the disease is very rarely cured except in cases where the general tuberculous process subsides." This statement is echoed by Ballenger and Ballenger [2]: "Generally speaking, the prognosis is unfavorable. There are, however, numerous exceptions to the rule." Lederer [3] stated: "The prognosis of middle ear tuberculosis should be guardedly based on the general condition of the patient as well as the nature of the underlying pathologic process. The progress of the pulmonary process is of paramount significance." The general opinion was well summed up by the statement of Myerson [4]: "If the chest lesion heals, the process in the ear is likely to heal."

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1. Phillips, W. C.: Diseases of the Ear, Nose, and Throat: Medical and Surgical, ed. 6, Philadelphia, F. A. Davis Company, 1922, p. 411.

2. Ballenger, W. L., and Ballenger, H. C.: Diseases of the Nose, Throat, and Ear: Medical and Surgical, ed. 7, Philadelphia, Lea & Febiger, 1938, p. 728.

3. Lederer, F. L.: Diseases of the Ear, Nose, and Throat: Principles and Practice of Otorhinolaryngology, ed. 4, Philadelphia, F. A. Davis Company, 1943, p. 156.

4. Myerson, M. C.: Tuberculosis of the Ear, Nose, and Throat, Including the Larynx, the Trachea, and the Bronchi, Springfield, Ill., Charles C Thomas, Publisher, 1944, p. 155.

Treatment of tuberculous otitis media other than that of the pulmonary lesion varied from a hands-off policy, through the use of various local medications and heliotherapy, to the employment of radical mastoidectomy. Most authorities, though, were in agreement that operation should be performed only when the complications of facial paralysis, meningitis or brain abscess occurred or threatened to occur, and then only if the general condition of the patient would warrant the procedure. Local treatment gave varying results in different hands. Cohen and Koepcke [5] reported 6.6 per cent healed perforations and 51.4 per cent apparently healed ears with the use of heliotherapy and the insufflation of a powder of 25 per cent potassium iodide in boric acid. They found that the ears apparently healed in some patients in spite of the fact that their general condition steadily grew worse and death ensued. In other patients the otitis media developed while the pulmonary lesion was improving, and the ear continued to discharge and did not heal, even though the pulmonary tuberculosis apparently was controlled. Jackson and Jackson [6] did not look favorably on any type of therapy, stating that "Tuberculous otitis media is not easily cured. The disease is usually chronic and mild. It resists treatment of any and all kinds."

The first reports on the use of streptomycin in otitic infections were concerned with its employment in nontuberculous infections. While the sulfonamides and penicillin had given excellent results in the majority of cases of purulent otitis media, they were ineffective in those instances in which the organism was either the gram-negative Bacillus proteus or Pseudomonas aeruginosa. Holt and Snell [7] reported on the use of streptomycin by the intramuscular route and by local instillation in 20 cases and obtained complete drying of 50 per cent of the ears and improvement in an additional 31 per cent. Pulaski and Matthews [8] found that in 13 cases of chronic otitis media there was benefit in 11 by the instillation of streptomycin. Harris, Kilham and Finland [9] treated 27 suppurating ears with local administration of streptomycin, and in their series, 15 ears became and remained dry; 7 improved, but relapse occurred, and 5 showed no improvement. In 14 ears resistant strains of

5. Cohen, S. S., and Koepcke, G. W.: Tuberculosis of the Ear and Mastoid in Adults, Am. Rev. Tuberc. **45**:427-436 (April) 1942.

6. Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat, and Ear, Including Bronchoscopy and Esophagoscopy, Philadelphia, W. B. Saunders Company, 1945, p. 402.

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9. Harris, H. W.; Kilham, L., and Finland, M.: Local Streptomycin Therapy for Suppurative Otitis, Arch. Otolaryng. **49**:69-77 (Jan.) 1949.

organisms developed, i. e. those organisms not inhibited by 50,000 units of streptomycin in each cubic centimeter of culture medium. Lundon [10] obtained "dry" ears in 15 out of 20 cases with treatment by the topical instillation of 5,000 micrograms of streptomycin in 1 cc. of saline solution twice daily over a period of two weeks, and he stated the belief that this method had a definite place in the treatment of chronic suppurative otitis media.

A review of the literature shows only two reports on the use of streptomycin in tuberculous otitis media. Grief and Gould [11] obtained healing of the perforations in 2 cases of tuberculous otitis media, proved by culture, using streptomycin by the intramuscular route. In a third case the medication was given intramuscularly and locally, but the ear remained moist and acid-fast bacilli were present which were found to be resistant to a concentration of streptomycin of more than 10 units per cubic centimeter. In a fourth case of bilateral tuberculous otitis media and mastoiditis, bilateral mastoidectomy was performed and streptomycin was given, both intramuscularly and instilled into the mastoid cavities, with healing of the wounds and tympanic membranes. Adams [12] reported 2 cases of tuberculous otitis media. In 1, the condition became quiescent after nearly two years of conservative therapy, consisting in cleansing of the canal and instillation of boric acid and alcohol drops. In a second case, in which the organisms were found on both smear and culture, the ear became dry in one month and remained dry after the use of 2 Gm. of streptomycin daily for one hundred and twenty days. Gilbert, Aronoff and Rosen [13] reported 9 cases of tuberculous otitis media treated with streptomycin, but they could not draw any definite conclusions from their series.

The present study is based on 36 patients with tuberculous otitis media who have been treated in various (sixteen) hospitals of the Veterans Administration (table), 6 of whom I treated. Streptomycin was used by parenteral administration in 32 patients and was instilled locally in 4. In the former group, the right ear was involved in 16 patients and the left ear in 13; involvement was bilateral in 3. In these 3, the ears were equally affected. The duration of the discharge varied from six years to one day. All these patients had active pulmonary tuberculosis, and acid-fast bacilli were found in the discharge by smear,

10. Lundon, A. E.: Streptomycin by Topical Application in Treatment of Chronic Suppurative Otitis Media, Arch. Otolaryng. **48**:418-429 (Oct.) 1948.

11. Grief, J. L., and Gould, J. W.: Tuberculosis of the Ear, Nose, and Throat Treated with Streptomycin, Arch. Otolaryng. **48**:209-220 (Aug.) 1948.

12. Adams, J. G.: Tuberculosis of the Middle Ear and Temporal Bone, Ann. Otol., Rhin. & Laryng. **57**:980-983 (Dec.) 1948.

13. Gilbert, J. G.; Aronoff, J. S., and Rosen, D. M.: Streptomycin Therapy in Otolaryngic Tuberculosis, Quart. Bull., Sea View Hosp. **10**:3-26 (Jan.) 1948.

culture or guinea pig inoculation in 29 patients (82.8 per cent). In the remainder, diagnosis was made on the basis of a painless aural discharge occurring in a case of pulmonary tuberculosis with positive sputum. The amount of streptomycin used varied, since in the majority of instances therapy was directed toward the pulmonary involvement.

*Data on 36 Patients with Tuberculous Otitis Media Treated with Streptomycin*

| Case No. | Ear | Duration of Discharge | Acid-Fast Bacilli | Dosage of Streptomycin | Result |
|---|---|---|---|---|---|
| 1 | L | 3 wk. | Smear | 2 Gm. daily 90 days | Unimproved |
| 2 | L | 4 wk. | Smear | 0.5 Gm. daily 56 days | Healed |
| 3 | L | 10 days | None | 1.0 Gm. daily 42 days | Healed |
| 4 | L | 5 wk. | Smear | 1.0 Gm. daily 42 days | Healed |
| 5 | R | 6 years | Culture | 1.0 Gm. daily 120 days | Improved |
| 6 | L | 5 mo. | Smear; culture | 1.0 Gm. daily 9 months | Healed |
| 7 | L | 3 yr. | None | 1.0 Gm. daily 42 days | Improved |
| 8 | R | 3 mo. | Culture | 1.0 Gm. daily 42 days | Unimproved |
| 9 | L | 2 mo. | Smear | 1.0 Gm. daily 42 days | Improved |
| 10 | R | 1 day | Culture | 1.0 Gm. daily 120 days | Healed |
| 11 | R | 2 wk. | Culture | 1.0 Gm. daily 60 days | Improved |
| 12 | R | 1 mo. | None | 0.5 Gm. daily 90 days | Unimproved |
| 13 | L | 5 days | Culture | 1.8 Gm. daily 120 days | Improved |
| 14 | L | 1 yr. | None | 1.0 Gm. daily 70 days | Healed |
| 15 | R | 2 mo. | None | 1.0 Gm. daily 120 days | Improved |
| 16 | R | 3 mo. | Smear | 1.0 Gm. daily 120 days | Healed |
| 17 | B | 5 mo. | Smear | 1.8 Gm. daily 120 days | Healed |
| 18 | R<br>L | 20 mo.<br>1 mo. | Guinea pig culture | 1.0 Gm. daily 42 days | Healed |
| 19 | R | 2 mo. | Smear; culture | 2.0 Gm. daily 120 days | Healed |
| 20 | L | 3 mo. | Smear; culture | 2.0 Gm. daily 120 days | Healed |
| 21 | R | 2 days | Culture | 1.0 Gm. daily 42 days | Unimproved |
| 22 | R | 3 mo. | None | 1.0 Gm. daily 120 days | Unimproved |
| 23 | L | 2 mo. | Culture | 1.0 Gm. daily 46 days | Improved |
| 24 | R<br>L | 2 mo.<br>6 mo. | Smear<br>Smear | 2.0 Gm. daily 120 days<br>2.0 Gm. daily 120 days | Healed<br>Healed |
| 25 | L | 5 mo. | Smear | 2.0 Gm. daily 60 days | Improved |
| 26 | R | 3 mo. | Smear; culture | 0.5 Gm. daily 49 days | Unimproved |
| 27 | R | 2 mo. | Smear | 1.0 Gm. daily 42 days | Healed |
| 28 | R | 10 mo. | Culture | 2.0 Gm. daily 120 days | Healed |
| 29 | R | 10 mo. | Smear; culture | 1.0 Gm. daily 42 days | Healed |
| 30 | R | 1 mo. | Culture | 1.0 Gm. daily 42 days | Improved |
| 31 | L | 3 mo. | Culture | 1.0 Gm. daily 25 days | Improved |
| 32 | R | 1 mo. | Smear; culture | 0.5 Gm. daily 42 days | Improved |
| 33 | R | 4 mo. | Culture | 0.1 Gm. instilled 20 days | Improved |
| 34 | R | 1 wk. | Culture | 0.1 Gm. instilled 20 days | Improved |
| 35 | L | 6 wk. | Culture | 0.1 Gm. instilled 5 days | Improved |
| 36 | L | 10 days | Smear | 0.1 Gm. instilled 18 days | Healed |

Treatment of tuberculous otitis media with streptomycin yields better results than that with the methods previously employed. Of the cases in which streptomycin was administered parenterally, healing of the perforations in both tympanic membranes occurred in the 3 cases of bilateral involvement. In the remaining 29 ears, healing of the perforations resulted in 12; 11 showed improvement in that the discharge ceased and did not recur during the time the patient was under

observation, but the perforation remained open. In 6 no improvement was noted, in that the discharge persisted during and after treatment.

In the cases in which streptomycin was applied locally, the technic used was cleansing of the external canal with cotton applicators to remove all visible discharge. One cubic centimeter of solution containing 0.1 Gm. of streptomycin was instilled into the canal, and by means of a Siegel otoscope the medication was gently forced into the middle ear. Two grams of the drug were used in 3 patients, and 0.5 Gm. were used in a fourth, the discharge having ceased on the third application in this instance. The results by this method were uniformly good, the discharge ceasing and not reappearing in 3 and the perforation healing in 1.

## SUMMARY

Streptomycin was used parenterally in 32 patients with tuberculous otitis media involving 35 ears, resulting in healing of the perforations in 18 ears (51 per cent); improvement in 11 (31 per cent), and no improvement in 6 (17 per cent). The drug was instilled into the middle ear in the remaining 4 patients, with improvement in 3 and healing of the perforation in the drum membrane in 1.

This study could not have been completed without the aid and assistance of Arthur M. Walker, M.D., Secretary of the Committee on Streptomycin, and the staffs of the Veterans Administration Hospitals at Brecksville, Ohio; Castle Point, N. Y.; Dayton, Ohio; Excelsior Springs, Mo.; Hines, Ill.; Legion, Texas; Richmond, Va.; Van Nuys, Calif.; Walla Walla, Wash.; Minneapolis, Minn.; Outwood, Ky.; Springfield, Mo.; Sunmount, N. Y.; Martinsburg, W. Va.; Tucson, Ariz., and the Glen Lake Sanatorium, Oak Terrace, Minn.

# DIAGNOSIS OF INTRACRANIAL LESIONS BY VESTIBULAR TESTS

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THE BASIS of this paper is the study of 30 patients with intracranial lesions, mostly tumors, confirmed at operation or autopsy, for whom a vestibular test was made previous to the surgical procedure. The patients were seen in the period from June 15, 1948 to June 15, 1949, during my residency in the service of otolaryngology at the Graduate Hospital of the University of Pennsylvania. Most of the patients were referred to the department by Dr. Robert A. Groff and Dr. Joseph C. Yaskin, chiefs of the departments of neurosurgery and neurology.

All the patients presented here were neurologically examined and studied completely, and it is believed that in many instances a great deal of reliance was placed on the findings of the vestibular tests. It is desired to emphasize that while the vestibular tests are of great importance in making a diagnosis of brain lesion, and occasionally may be the only sign pointing to a lesion, complete studies and neurologic examination are necessary. Most frequently the vestibular findings must be correlated with all the other findings before the important decision to explore the brain is made. Again, the vestibular findings, on occasion, may be of such importance as to change the location of search by the neurosurgeon from the anterior part of the skull to the posterior. However, it must be stressed that valuable as the vestibular tests are they still must be regarded as but one of the important tools of diagnosis of intracranial lesions. In many cases it was found that the vestibular tests did not indicate a lesion, whereas neurologically the findings justified the exploration of the brain.

## PRELIMINARY STUDY

*History.*—Only a brief history was taken, since all the patients seen were referred from other services, in which a complete history had

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already been taken. The symptoms of the 30 patients are presented in table 1.

*Otolaryngologic Examination.*—An otolaryngologic examination was performed in all cases. It is of special importance to examine both ears carefully and to look for such things as cerumen and perforations and to be certain that there will be nothing to interfere with the test. Tuning fork tests were made for all the patients, and in some cases audiometric studies were made. It is extremely important to make a complete hearing evaluation (voice, tuning fork, audiometry and speech tests), because failure to do so might compromise the correct diagnosis, as will be shown in case 25.

TABLE 1.—*Symptoms of 30 Patients with Intracranial Lesions*

| Symptoms | Infratentorial Tumors (10) | Supratentorial Lesions (13) | Cerebellopontile Angle Tumors (7) |
|---|---|---|---|
| Headaches | 10 | 10 | 3 |
| Vomiting | 10 | 3 | 1 |
| Deafness | 1 | 2 | 7 |
| Staggering | 9 | 1 | 6 |
| Dizziness | 4 | 1 | 2 |
| Weakness | 0 | 6 | 2 |
| Blurring of vision | 3 | 3 | 2 |
| Double vision | 0 | 0 | 2 |
| Tinnitus | 0 | 2 | 4 |
| Convulsions | 0 | 3 | 0 |
| Paresthesias | 0 | 1 | 4 |
| Aphasia | 0 | 3 | 0 |

## VESTIBULAR TESTS

The vestibular studies were carried out in the following order.

A. Spontaneous phenomena were looked for: spontaneous nystagmus, postural nystagmus, spontaneous vertigo, tendency toward falling and spontaneous past pointing.

B. Caloric or rotation tests were performed, but galvanic tests were not made, for it was believed that their usefulness is greater in peripheral lesions in differentiating labyrinthine lesions from those of the eighth nerve.

A. *Spontaneous Phenomena.*—Spontaneous Nystagmus: It is known that spontaneous nystagmus is not present normally. The determination of spontaneous nystagmus and its character is very important, because it must not be confused with the nystagmus elicited after the stimulation of the labyrinth. Spontaneous nystagmus is relatively a common finding in intracranial lesions. In Nylén's study of 673 patients with brain tumors, he found nystagmus present in 57 per cent. Of the 30 patients in this series, spontaneous nystagmus was present in 12. It is of some interest to note that it was found in 6 out of 10 patients with

verified infratentorial tumors and in 6 out of 7 with tumors of the acoustic nerve; but spontaneous nystagmus was not present in the 13 patients with proved supratentorial lesions. In testing for spontaneous nystagmus it should not be confused with ocular nystagmus. The main features which were of help in differentiating the two are presented in table 2. These two types of nystagmus can also be differentiated by optic nystagmus tests, such as the Brunner umbrella test. The responses to the optic nystagmus test are shown in table 3.

Postural Nystagmus: The postural or positional nystagmus tests were made in some cases but not in a number sufficient for comment on

TABLE 2.—*Features for Differentiation of Vestibular and Ocular Nystagmus*

| Vestibular | Ocular |
|---|---|
| Rhythmic, coordinated movement of both eyes, with a slow and quick component | Irregular oscillation of the eyes, without the slow and quick component |
| (Peripheral type)<br>Mixed: rotary and horizontal, of short duration, with tendency to disappear; history of labyrinthine or cochlear disturbances | Generally of long duration; usually no complaint of this symptom by patient |
| | Sometimes patient able to stop the nystagmus voluntarily |
| (Central type)<br>Pure: vertical, of long duration, with tendency to progress, accompanied with neurologic symptoms and signs | Nystagmus concomitant with ocular pathologic conditions |

TABLE 3.—*Responses to the Optic Nystagmus Test*

| Normal Person | Patient with Spontaneous Nystagmus of Vestibular Origin | Patient with Spontaneous Nystagmus of Ocular Origin |
|---|---|---|
| Rotation of the cylinder to the right elicits nystagmus to the left, and vice versa | Response of such a patient will be exactly as that of a normal person | Rotation of the cylinder to the right elicits nystagmus to the right, and vice versa; this test might stop the nystagmus |

their value. These tests, however, are deemed to be of considerable importance. Of Nylén's 673 patients with brain tumors, 57 per cent showed spontaneous nystagmus, and he pointed out that 80 per cent showed nystagmus after positioning.

Spontaneous Vertigo: Many patients in this series complained of vertigo. However, most often in cases of intracranial lesions, the patients' interpretation of vertigo was that of unsteadiness. When the vertigo was rotational, it was most often due to a peripheral irritation (labyrinthine) rather than to a central lesion.

Tendency Toward Falling: The Romberg test was done on all the patients except those unable to stand, and a positive sign was elicited from many; a history of a tendency to fall was searched for and found

in many cases. The pelvic girdle test, or attempt to overthrow, was also made and poor resistance was often found in patients with intracranial lesions, especially in those with infratentorial and cerebellopontile angle tumors.

Past Pointing: When past pointing existed spontaneously and could not be modified by the vestibular stimulation, an organic cerebellar lesion was expected. Spontaneous past pointing was oftener found in cases of cerebellopontile angle tumors.

The table of spontaneous findings (table 4) was of some value in the differentiation of peripheral and central lesions, but the table is only schematic and therefore should not be regarded as conclusive.

B. *Caloric or Rotation Vestibular Tests.*—The mass caloric irrigation, rotation test and minimal caloric stimulation of Kobrak were the only tests routinely performed, because it was felt that other tests used

TABLE 4.—*Spontaneous Findings on the Basis of Peripheral and Central Lesions*

| Findings | Peripheral Lesions | Central Lesions |
|---|---|---|
| Spontaneous nystagmus | Mixed type: short duration; accompanied with labyrinthine and cochlear disturbances | Pure type: long duration; accompanied with neurologic symptoms and signs |
| Postural nystagmus | Always toward the same direction, regardless of position of body or head | Direction changes with change in position of body or head |
| Spontaneous vertigo | Usually associated with tinnitus and deafness | Usually accompanied with good hearing and loss of consciousness |
| Tendency toward falling | In the direction opposite that of the nystagmus; direction of the falling changes when position of the head is changed | In the same direction as that of the nystagmus; direction not changed with change in position of head |
| Past pointing | In the direction opposite that of the nystagmus | In the same direction as that of the nystagmus |

to study the function of the vestibular apparatus were not sufficient for the purpose of determining the presence of intracranial lesions.

The rotation test was used only when mass caloric irrigation was contraindicated (recent perforations, etc.). The rotation test does not disturb the patient as much as the mass caloric test, but in the rotation test both labyrinths are tested at the same time.

Kobrak's test of minimal caloric stimulation, with slight modifications, was performed with several patients. It does not disturb the patient much, and each labyrinth can be tested separately, but in my hands the study of vertigo, the tendency toward falling and past pointing was not satisfactory with this test.

Mass caloric irrigation with cold water at 68 F., or 20 C., was thus preferred, because each ear could be stimulated and hence examined separately. In addition, the vertical and horizontal canals could be studied individually, and the study of the vertigo, tendency toward falling and past pointing was satisfactory. It is true that in some patients

there developed more or less violent reactions of the sympathetic nervous system, such as pallor, perspiration, nausea and vomiting; but is also true that these unpleasant reactions were useful, in many instances, in determining whether a lesion was above or below the tentorium.

Mass caloric irrigation with cold water was the vestibular test most frequently used during my residency at the Graduate Hospital, where more than 400 vestibular tests were made in a one year period. Following is a description of the procedure of and the normal responses to this vestibular test.

To test the vertical canal, the patient's head is placed 30 degrees forward and the right ear is douched from forty-five to sixty seconds. Rotatory nystagmus occurs to the left; past pointing, to the right, from 3 to 8 inches (7.6 to 20 cm.), and tendency to fall to the right.

Immediately after these reactions are quickly noted, the head is placed 60 degrees backward to test the horizontal canal. Horizontal nystagmus occurs to the left; past pointing, to the right, from 3 to 8 inches, and the tendency to fall to the right. Indications of sensitivity to the test are pallor, perspiration and nausea (none very marked).

The patient is allowed a short rest period, and the left ear is then tested.

To test the vertical canal, the head is placed 30 degrees forward. After the left ear is douched for forty-five to sixty seconds, the following reactions occur. Rotary nystagmus occurs to the right; past pointing, to the left, from 3 to 8 inches (7.5 to 20 cm.) and tendency to fall, to the left.

Immediately after these reactions, the head is placed 60 degrees backward to test the horizontal canal.

Horizontal nystagmus occurs to the right; past pointing, to the left, from 3 to 8 inches, and tendency to fall, to the left. The indications of sensitivity to this test are the same as that after the right ear is douched.

In many cases it was found that after the ear was douched the responses of the horizontal canals were more pronounced than those of the vertical canals in patients for whom all the vestibular findings were otherwise within normal limits.

### INTERPRETATION OF VESTIBULAR TESTS

It is known that abnormal vestibular responses are seen in various conditions involving the labyrinth, the vestibular nerve, the vestibular center, or the center pathways (i. e., Deiters' nucleus, medial longitudinal bundle, cerebellum, etc.).

In the cases reported in this paper, a central lesion was suspected if "central spontaneous findings" were present (see table 4). If, after the vestibular stimulation, instead of a normal nystagmus, the nystagmus

was perverted, inverted, mixed, exaggerated or with conjugate deviation or nystagmoid movements, a central lesion was suspected.

Central lesions were also suspected when normal responses were obtained from the canals in one plane and none or abnormal responses from the canals of the other plane.

A persistent and definite past pointing which was present spontaneously and was not altered by the vestibular stimulation was suggestive of a central lesion.

A central lesion was suspected if nystagmus was present but vertigo or past pointing absent, or if the situation was reversed.

If dissociated nystagmus was seen, the probability of central lesion was considered. It was difficult to establish a diagnosis based only on the hypoactivity or hyperactivity of the responses on caloric stimulation, unless other vestibular responses were present.

### LOCATION OF CENTRAL LESIONS

After the presence of a central lesion was established, the location of the lesion was sought. On the basis of vestibular tests, intracranial lesions may be divided into three main groups: infratentorial, supratentorial and cerebellopontile angle.

*Infratentorial Lesions.*—It was suspected that the lesion was localized below the tentorium (posterior cranial fossa lesion) because of the following factors.

1. All the patients in this study had good hearing except 1 (case 10), who had some hearing loss. Audiometric studies revealed an approximate loss of 8.7 per cent for the left ear and 14.4 per cent for the right mainly in the high tone range, but this particular patient was 56 years old and his hearing loss was considered due to presbycusis. Hearing is usually not impaired unless the lesion in the posterior cranial fossa is secondary to a labyrinthitis or is located in the cerebellopontile angle.

2. Poor movement of the pelvic girdle was present in the patients tested for it and was probably present in those patients for whom it was difficult to make this test, the difficulty being due to the age of some of them, 3, 3½, 5 and 7½ years.

3. No sensitivity to the test, or very little, was found in all the patients with infratentorial lesions. I believe that the sensitivity of the patient to the test is a very important factor to consider. Shuster stressed the fact that most infratentorial lesions are accompanied with little or no sensitivity. On the other hand, most of the supratentorial lesions are accompanied with high sensitivity, which was found to a remarkable extent in most of the patients in this study. However, sensitivity occasionally may be absent or mild in patients with lesions of the occipital or temporal lobes.

4. Disturbances of equilibrium were found in many patients, as well as disturbances of coordination, such as inability to touch the finger to the nose and adiadokokinesis.

5. Vertigo, after stimulation of the labyrinth, was not pronounced in most of the patients and, in fact, was markedly diminished on many occasions.

6. Each side of the cerebellum controls muscular coordinating functions on that side of the body. Interference with muscular coordinating functions on one side (abnormal vestibular responses) suggests that the lesion is situated on that side. If, however, both sides show impaired vestibular responses, the lesion is most probably located close to the midline.

Ten of the 30 patients in this series had infratentorial tumors confirmed at operation or autopsy.

*Supratentorial Lesions.*—In the 13 patients with supratentorial lesions, the responses of the vestibular apparatus were more difficult to evaluate. In most cases it was possible only to localize the lesion above the tentorium and to determine whether it was inclined to the right or to the left. The following data might be helpful for localization of a lesion in the parieto-occipital-temporal lobes.

1. Impaired vestibular responses from one ear (right ear)

2. Good hearing on the same side (right ear)

3. Impaired responses from the vertical canal of the opposite ear (left ear)

4. High sensitivity to the vestibular test

5. Usually good resistance to the pelvic girdle test

6. Sufficient evidence of spontaneous findings referable to the central nervous system (table 4).

If a supratentorial lesion is suspected, and the impairment exists in the vertical canal, the lesion would most likely be on the side opposite that from which the impaired responses were elicited. But if the impairment is found in the horizontal canal, the lesion would most probably be on the same side from which the impaired responses were obtained.

In cases of lesions of the pituitary gland the vertigo is usually of shorter duration than the nystagmus. Sensitivity may or may not be absent. Other signs of a central lesion are present spontaneously and after stimulation.

In cases of lesions of the suprasellar region there might be exaggerated nystagmus out of proportion to the vertigo. Usually present are impaired responses from the vertical canal of one side and poor resistance to the pelvic girdle test.

In cases of lesions of the occipital lobe there may be a loss of responses to stimulation; perverted responses from the horizontal canals, and, usually, prolonged and exaggerated past pointing out of proportion to the vertigo.

*Cerebellopontile Angle Lesions.*—I believe that the vestibular examination is of paramount importance in the early diagnosis of tumors of the acoustic nerve before the appearance of advanced symptoms and at a time that surgical treatment promises the best results. For most of the 7 patients with cerebellopontile angle lesions, the vestibular tests showed the typical "Bárány picture" of a tumor of the acoustic nerve characterized by the following signs.

1. Loss of hearing in one side (right ear)
2. Loss of vestibular responses on the same side (right ear)
3. Impaired responses from the vertical canal of the opposite side (left ear)
4. Little or no sensitivity to the vestibular test
5. Occasionally perverted responses from the horizontal canal of the opposite ear (left ear)
6. Presence of spontaneous findings referable to the central nervous system
7. Presence of the lesion on the same side from which cochlear and vestibular responses were absent.

SUMMARY

The importance and usefulness of the vestibular tests as an aid in the diagnosis and localization of intracranial lesions are emphasized. It is also stressed that the vestibular findings must be correlated with all the data obtained by other examinations before a definite conclusion is arrived at and before the important decision is made to explore the brain.

The technic for the procedure of the mass caloric vestibular tests is briefly described and the interpretation of the findings is discussed.

The protocols of 30 extensive case reports are presented (10 of infratentorial tumors; 13 of supratentorial lesions, and 7 of cerebellopontile angle tumors). In the 30 cases the diagnosis was confirmed at operation or autopsy, and in all the patients studied a vestibular examination was done previous to the surgical procedure, the results of which had been mostly in accord with the postoperative or postmortem findings.

13 Number 154, Vedado.

Dr. Edward H. Campbell and Dr. George M. Coates, Chiefs of the Service of Otology, and Dr. Benjamin H. Shuster, in charge of Neuro-Otology, afforded me the opportunity to make the vestibular tests and to study their patients.

## APPENDIX

### PROTOCOLS OF CASES OF INFRATENTORIAL LESIONS

CASE 1.—Girl, 7½ years old, admitted on Jan. 17, 1949

*History.*—Vomiting, generalized headaches of 1½ years' duration; lately, some staggering

*Examinations.*—Neurologic: Markedly ataxic gait, reflexes generally depressed; sleepiness

Ophthalmologic: Bilateral papilledema and hemorrhages

Roentgenologic: Evidences of increased intracranial pressure

*Vestibular Tests.*—Diagnosis based on the perverted nystagmus from both vertical canals; perverted nystagmus from the horizontal canal of the left ear; no sensitivity to the tests; good hearing: "suspected lesion of the posterior cranial fossa, midline"

*Operation.*—Midline tumor in the cerebellum (Dr. Groff)

*Microscopic Diagnosis.*—Gliofibrillary medulloblastoma

CASE 2.—Boy, 5 years old, admitted on March 1, 1949

*History.*—Bifrontal headaches, dizziness, vomiting, slight difficulty in walking; symptoms of five months' duration

*Examinations.*—Neurologic: Slight truncal ataxia; questionably positive Romberg sign

Ophthalmologic: No papilledema; "picture more like pseudoneuritis"

*Vestibular Tests.*—Diagnosis based on perverted nystagmus from both horizontal canals, no sensitivity, good hearing: "suspected lesion of the posterior cranial fossa, midline" (March 3, 1949)

*Ventriculogram.*—Done five days later; changes consistent with a diagnosis of a lesion of the posterior cranial fossa

*Operation.*—Large infiltrating cerebellar tumor in the midline (Dr. Groff)

*Microscopic Diagnosis.*—Fibrillary astrocytoma

CASE 3.—Man, 27 years old, admitted on Jan. 17, 1949

*History.*—Vomiting, generalized headaches, dizziness, staggering; lately, some blurring of vision; symptoms of four and one half years' duration; 35 pound loss of weight (15.5 Kg.) in last four months

*Examinations.*—Neurologic: Slightly ataxic gait with eyes closed; corneal sensitivity on the left slightly less; depressed tendon reflexes

Ophthalmologic: Bilateral papilledema; normal visual fields

*Vestibular Tests.*—Diagnosis based on spontaneous horizontal and vertical nystagmus (also reported by other services); poor resistance to pelvic girdle test; perverted and inverted responses from the vertical canal of the right ear; perverted responses from the vertical canal of the left ear; perverted and inverted responses from the horizontal canal of the right ear; no sensitivity; good hearing: "suspected lesion of the posterior cranial fossa, midline"

*Operation.*—Midline cerebellar tumor (Dr. Schuerman)

*Microscopic Diagnosis.*—Ependymoma

CASE 4.—Woman, 28 years old, admitted on Feb. 25, 1949

*History.*—Frontal headaches, nausea, vomiting, dizziness, diplopia, of ten months' duration; lately pronounced staggering

*Examinations.*—Neurologic: Staggering gait; bilateral dyssynergia; peripheral weakness of the sixth, the seventh and the eleventh cranial nerve, on the left; positive Romberg sign

Ophthalmologic: Bilateral papilledema; spontaneous nystagmus; enlarged blind spots bilaterally

Roentgenologic: Chest, normal; skull, changes consistent with increased intracranial pressure

*Vestibular Tests.*—Diagnosis based on spontaneous vertical and horizontal nystagmus; poor pelvic girdle movement; perverted responses from the horizontal canal of the right ear and the vertical canal of the left ear; perverted and inverted responses from the horizontal canal of the left ear; no sensitivity; good hearing: "suspected lesion of the posterior cranial fossa, midline"

*Operation.*—Midline cerebellar tumor (Dr. Groff)

*Microscopic Diagnosis.*—Medulloblastoma

Case 5.—Boy, 3½ years old, admitted on June 21, 1948

*History.*—Normal until patient 6 weeks old; then began to tilt head to the right; left eye turned in five months later; vomiting, two years later; recently began to stagger; became uncoordinative; intense headaches in the occipital region 2 days ago

*Examinations.*—Neurologic: Spontaneous nystagmus; paralysis of the sixth cranial nerve on the left; questionable central paralysis of the seventh cranial nerve; positive Romberg sign (falling to the left); ataxic gait; bilateral dyssynergia; diminished tendon reflexes; definite "cracked pot" sign

Ophthalmologic: Bilateral papilledema; spontaneous nystagmus

Roentgenologic: Chest, normal; skull, changes due to increased intracranial pressure

*Vestibular Tests.*—Diagnosis based on the horizontal and vertical spontaneous nystagmus; tendency to fall to the left; absence of responses from both canals of the right ear; no sensitivity; good hearing: "suspected lesions of the posterior cranial fossa toward the right side" (June 22, 1948)

*Ventriculogram.*—Done eight days later; changes regarded as indicative of a lesion mass in the region of the third ventricle

*Operation.*—Midline cerebellar tumor (Dr. Groff)

*Microscopic Diagnosis.*—Astrocytoma, mildly fibrillary, type 2

Case 6.—Boy, 3 years old, admitted on April 25, 1949

*History.*—Headaches, vomiting, staggering, suppurative otitis media in left ear (clear, colorless fluid); symptoms of three months' duration

*Examinations.*—Neurologic: Depressed reflexes; positive Babinsky sign bilaterally; questionable nuchal rigidity

Ophthalmologic: Mild degree of convergence; bilateral papilledema

Roentgenologic: Chest and mastoid processes, normal; evidences of increased intracranial pressure

*Vestibular Tests.*—Diagnosis based on the perverted responses from all canals of both ears, no sensitivity, good hearing: "suspected infratentorial lesion of midline"

*Operation.*—Midline cerebellar tumor (Dr. Groff)

*Microscopic Diagnosis.*—Neuroblastoma

CASE 7.—Man, 21 years old, admitted Feb. 19, 1949

*History.*—Severe headache lasting two or three minutes, of three weeks' duration; on admission, headaches accompanied by vomiting and staggering

*Examination.*—Neurologic: Marked personality changes, irritability, uncooperativeness; slight dyssynergia on the left; spontaneous nystagmus

Ophthalmologic: Early papilledema, more pronounced in the left eye

Roentgenologic: Chest and skull, essentially normal

*Vestibular Tests.*—Diagnosis based on the horizontal and vertical spontaneous nystagmus; poor resistance to pelvic girdle test; perverted responses from both horizontal canals; no sensitivity; good hearing: "suspected lesion of posterior cranial fossa, near midline" (Feb. 22, 1949)

*Ventriculogram.*—Done two days later; suggestive of lesion of the posterior cranial fossa

*Operation.*—Tumor in the right cerebellar hemisphere (Dr. Schuerman)

*Microscopic Diagnosis.*—Medulloblastoma

CASE 8.—Girl, 17 years old, admitted on Jan. 29, 1949

*History.*—Headaches of six months' duration; nausea and vomiting on only two occasions; lately, blurring of vision

*Examinations.*—Neurologic: Some adiadokokinesis of the left hand; decided ataxic gait; veering toward the left; all reflexes diminished

Ophthalmologic: Bilateral papilledema; appearance of the left fundus very suggestive of von Hipple–Lindau disease, but bilaterality of the process against this

Roentgenologic: Chest and skull, essentially normal

*Vestibular Tests.*—Diagnosis based on poor resistance to pelvic girdle test; perverted responses from the horizontal canal of the right ear and from the vertical canal of the left ear; no sensitivity; good hearing: "suspected infratentorial lesion, midline" (Feb. 1, 1949)

*Ventriculogram.*—Done the next day; changes regarded as indicative of a lesion mass on the posterior cranial fossa

*Operation.*—Tumor in the left cerebellar hemisphere (Dr. Groff)

*Microscopic Diagnosis.*—Astrocytoma, predominantly protoplasmic, type 1

CASE 9.—Woman, 34 years old, admitted on July 21, 1948

*History.*—Occipital headaches of four weeks' duration; unsteadiness of gait; tendency to fall to the right; several days of vomiting two weeks before admission; disappearance of vomiting at time of admission

*Examinations.*—Neurologic: Spontaneous nystagmus; dyssynergia in arms and legs; pronounced ataxic gait; positive Romberg sign (falling to the right); Hoffmann positive sign on the left; ankle clonus; hyperactive reflexes; pain in the neck and occipital region, with definite stiffness

Ophthalmologic: Normal eyegrounds; visual fields full

Roentgenologic: Skull, normal

*Vestibular Tests.*—Diagnosis based on the horizontal spontaneous nystagmus; spontaneous past pointing; tendency toward falling to the right; poor resistance to pelvic girdle test; decided past pointing after the douching (72 inches [183 cm.] with the left hand, 75 inches [190 cm.] with the right); no sensitivity; good hearing: "suspected lesion of the posterior cranial fossa, midline"

*Operation.*—Cerebellar tumor of the left lobe (Dr. Groff)

*Microscopic Diagnosis.*—Carcinoma, appearance metastatic

*Autopsy.*—Entirely encapsulated tumor (4 by 2.5 cm.) found in the cerebellum

CASE 10.—Man, 56 years old, admitted on Oct. 22, 1948

*History.*—Nausea and vomiting, of two months' duration; lately disappeared; recently unable to walk without assistance; numbness in the right arm

*Examination.*—Neurologic: Slight dyssynergia; weakness of the left arm and of both legs; increased reflexes on the left side; positive Hoffmann sign on the left; ataxic gait, positive Romberg sign; spontaneous nystagmus

Ophthalmologic: Normal eyegrounds; spontaneous nystagmus

Roentgenologic: Skull, essentially normal

*Vestibular Tests.*—Diagnosis based on the horizontal and vertical spontaneous nystagmus; tendency toward falling; poor resistance to pelvic girdle test; perverted responses from both horizontal canals; hearing loss questionable because of age (presbycusis); no sensitivity: "suspected lesion of the posterior cranial fossa near midline"

*Operation.*—Suboccipital craniectomy; posterior cranial fossa carefully explored; diagnosis, "possible intramedullary tumor of medulla, pons and upper cord" (Dr. Schuerman)

*Autopsy.*—Death one month later; pontile glioma found extending downward to the medulla and upper part of cord

PROTOCOLS OF CASES OF SUPRATENTORIAL LESIONS

CASE 11.—Woman, 29 years old, admitted on May 5, 1949

*History.*—Occasional, generalized convulsions, periods of aphasia and disorientation, of six years' duration; last seizure two weeks ago, without recovery

*Examinations.*—Neurologic: Mixed aphasia, more motor than sensory; mild central weakness of the right side of the face; weakness of the right arm; increased reflexes on right side; lack of sense of position; stereognosis in the right arm and hand; lack of coordination in the right arm

Ophthalmologic: "No gross ophthalmic lesion suspected on the basis of this incomplete examination"

Roentgenologic: Chest and skull, essentially normal

*Vestibular Tests.*—Diagnosis based on poor resistance to pelvic girdle test; perverted responses from both canals of the left ear; pronounced sensitivity; good hearing: "suspected supratentorial lesion, left side"

*Operation.*—Lesion mass very deeply situated under the motor area; no attempt to remove tumor because of location (Dr. Schuerman)

CASE 12.—Man, 48 years old, admitted on Nov. 5, 1948

*History.*—Headaches for indefinite period; jacksonian seizure on the left six weeks before admission; weakness in the left arm and leg; recently motor aphasia and personality changes developed; patient left handed

*Examinations.*—Neurologic: Hemiparesis on the left; tendon reflexes increased on the left; ankle clonus on the left; tenderness to percussion on the right side of the head

Ophthalmologic: Bilateral papilledema; venous engorgement; hemorrhages

Roentgenologic: Skull, essentially normal

*Vestibular Tests.*—Diagnosis based on spontaneous past pointing with the right arm; tendency toward falling; perverted responses from the vertical canal of the left ear; pronounced sensitivity; good hearing: "suspected supratentorial lesion, right side"

*Operation.*—Tumor in the right frontal lobe (Dr. Schuerman)

*Microscopic Diagnosis.*—Glioblastoma, with invasion of the leptomeninges

CASE 13.—Woman, 54 years old, admitted on Oct. 18, 1948

*History.*—Occipital headaches, nausea and vomiting, of four weeks' duration; lately, beginning mental failure; development of ptosis of the right eye and weakness on the left side of the face.

*Examinations.*—Neurologic: Complete paralysis of the third nerve; weakness of the seventh nerve, on the left; hemiparesis on the left; increased reflexes: positive Hoffmann sign on the left

Roentgenologic: Skull, essentially normal

*Vestibular Tests.*—Diagnosis based on perverted responses from both horizontal canals; high sensitivity; impaired responses thought to be greater in the right horizontal canal: "suspected supratentorial lesion inclined to the right" (Oct. 19, 1948)

*Ventriculogram.*—Done next day; lesion mass indicated in the right temporo-frontoparietal region

*Operation.*—Tumor in the right temporal lobe (Dr. Groff)

*Microscopic Diagnosis.*—Astrocytoma, protoplasmic, type 2

CASE 14.—Man, 57 years old, admitted on Sept. 15, 1948

*History.*—Convulsive twitching of the left side of the face and headache of two hours' duration first noted two months ago; several recurrences; lately, generalized weakness and inability to move both legs and arms; 33 pounds (15 Kg.) loss of weight in last three months

*Examinations.*—Neurologic: Central weakness on the left side of the face; hemiplegic gait on the left; dysdiadokokinesis; astereognosis of the left hand; positive Romberg sign (falling to the right); loss of sense of position on the left; more active knee jerk on the left

Ophthalmologic: Normal eyegrounds; right pupil larger than the left

Roentgenologic and Stereoscopic: Skull, normal

*Vestibular Tests.*—Diagnosis based on spontaneous past pointing to the left; tendency to fall to the right; perverted responses from the vertical canal of the left ear; questionable impairment of the vertical canal of the right ear; high sensitivity; slight impairment of hearing: "suspected supratentorial lesion, right side" (Sept. 16, 1948)

*Ventriculogram.*—Done five days later; a large mass situated in the right occipito-parietal region

*Operation.*—Large tumor in the right parieto-occipital lobe (Dr. Groff)

*Microscopic Diagnosis.*—Carcinoma, metastatic (Sept. 19, 1949); on roentgenogram, three days later, large round shadow on the right lower lobe of the lung; probably representing bronchiogenic malignant process or pulmonary metastasis

CASE 15.—Man, 34 years old, admitted on March 5, 1949

*History.*—Blurring of vision, intermittent burning sensation in the back of the neck, vomiting, headaches, tinnitus in the right ear; symptoms of three months' duration; hypertensive condition

*Examinations.*—Neurologic: "Essentially negative results"

Ophthalmologic: Bilateral papilledema; homonymous hemianopsia on the left; "intracranial space-taking lesion involving the right optic tract"

Roentgenologic: Skull, essentially normal; chest, enlargement of the left ventricle consistent with hypertensive cardiovascular disease

*Vestibular Tests.*—Diagnosis based on the perverted responses from the horizontal canal of the right ear (oblique nystagmus); perverted responses from the vertical canal of the left ear; delayed responses from the other two canals; decided sensitivity: "suspected supratentorial lesion, inclined to the right" (March 7, 1949)

*Ventriculogram.*—Done two days later; multiple lesions; larger one located in the right occipitotemporal region

*Operation.*—"Brain tumor found; intraventricular, particularly in the right temporoparietal lobe" (Dr. Schuerman)

*Microscopic Diagnosis.*—Neuroblastoma

CASE 16.—Woman, 48 years old, admitted on May 5, 1949

*History.*—Generalized headaches, considerable increase in appetite, some disturbance in memory of ten months' duration; lately, generalized weakness

*Examinations.*—Neurologic: "Essentially negative results"

Ophthalmologic: Bilateral papilledema

Roentgenologic: Chest and skull, essentially normal

*Vestibular Tests.*—Patient very uncooperative; diagnosis based on the responses elicited from the vertical canal of the right ear—rotatory nystagmus to the left, of a very large amplitude, hyperactivity—and from the horizontal canal of the right ear—horizontal nystagmus to the left, of a large amplitude, very rapid, somewhat nystagmoid movements appearing only after ten seconds of douching; (pronounced sensitivity to the test, causing refusal of patient to have the test repeated on the left ear): "suspected supratentorial lesion" (May 6, 1949)

*Ventriculogram.*—Done four days later; lesion mass in the right frontal region

*Operation.*—Tumor in the right sphenoid ridge (Dr. Groff)

*Microscopic Diagnosis.*—Meningioma, type 2, variety 1

CASE 17.—Woman, 39 years old, admitted on March 10, 1949

*History.*—First jacksonian seizure with loss of consciousness and temporary paralysis of the right foot eight years ago; since then, daily convulsions not accompanied with loss of consciousness, partially controlled by anticonvulsants; lately, only two mild attacks weekly; sudden increase in frequency of attacks (about one every five minutes) one week ago

*Examinations.*—Neurologic: Hemiparesis on the right; frequent convulsions characterized by tonic contractions of the right leg and foot, less markedly so of the arm; aphasia during attacks; conjugate deviation of the eyes to the right

Ophthalmologic: Bilateral papilledema; conjugate deviation to the right

Roentgenologic: Skull, dorsum sellae and posterior clinoid process poorly visualized; demineralized appearance of these bony structures; possibility of bone destruction

*Vestibular Tests.*—Diagnosis based on the conjugate deviation; perverted responses from the vertical canal of the right ear; perverted responses from both canals of the left ear; some sensitivity; good hearing: "suspected supratentorial lesion, left side"

*Operation.*—Left frontoparietal brain tumor (Dr. Groff)

*Microscopic Diagnosis.*—Oligodendroglioma

CASE 18.—Man, 45 years old, admitted on May 23, 1949

*History.*—Weakness of the right side of the body and aphasia, of seven weeks' duration

*Examinations.*—Neurologic: Motor aphasia; central weakness on the right side of the face; hemiparesis on the right; increased tendon reflexes on the right; positive Babinsky sign; astereognosis of the left arm; hypesthesia of the cornea on the right; some mental obtundity

Ophthalmologic: Normal except for decreased corneal sensation of the right eye

Roentgenologic: Chest and skull, essentially normal

*Vestibular Tests.*—Diagnosis based on the inverted responses from the vertical canal of the right ear; high sensitivity; good hearing: "suspected supratentorial lesion, inclined to the left"

*Operation.*—Left frontotemporal craniotomy (Dr. Shenkin); no tumor found; report of Dr. Shenkin: "fairly satisfied that there is a deep-lying lesion in the left temporal hemisphere." Patient discharged on June 18, 1949; kept under close observation

CASE 19.—Girl, 8 years old, admitted on July 28, 1948

*History.*—Intermittent headaches and tinnitus of three weeks' duration; facial motor asymmetry noticed by mother one week ago; complaint of black spots in front of eyes; tremendous weight gain in the past winter

*Examinations.*—Neurologic: Essentially normal results except for a central weakness on the left side of the face

Ophthalmologic: Bilateral papilledema; very enlarged blindspot in visual fields; nasal cut in the right eye

Roentgenologic: Skull, small calcification over the sella turcica; anterior clinoid processes definitely eroded

*Vestibular Tests.*—Diagnosis based on the perverted responses from the horizontal canal of the left ear; high sensitivity; good hearing: "suspected supratentorial lesion, left side." Suspicion that the lesion was on the left side based on impaired vestibular responses found in the horizontal canal on the left; impairment in the left vertical canal would have influenced localization of the lesion to the right side

*Operation.*—Large tumor, glioma, involving the entire optic chiasm; no biopsy taken—pathologic diagnosis clear from the appearance of the lesion and good fields of vision

CASE 20.—Woman, 33 years old, admitted on June 1, 1949

*History.*—Protrusion of the right eye; frontal headaches; no pain in the eye or visual changes; symptoms of two years' duration

*Examinations.*—Neurologic: Proptosis of the right eye; slight central weakness of the seventh cranial nerve on the left; positive Hoffmann sign on the left; questionable Babinsky sign; increased reflexes; slight blurring on the nasal border of the disks; "meningioma of the sphenoid ridge"

Ophthalmologic: "Retrobulbar mass of the right eye, possibly a mucocele of the right frontal sinus, neurofibroma of the ethmoid sinus or meningioma of the sphenoid ridge, lateral location"

Roentgenologic: Chest, normal; skull, irregular mass of bony density occupying the outer thirds of sphenoid ridge on the right, indicative of meningioma of the sphenoid ridge (first roentgenogram); additional films taken two days later; lesion within the right frontal sinus and extending down into the right orbit; appearance of the lesion characteristic of an osteoma of the frontal sinus rather than a meningioma

*Vestibular Tests.*—Diagnosis based on the perverted responses from the horizontal canal of the right ear and from both canals of the left ear; high sensitivity; good hearing: "suspected supratentorial lesion, right side"

*Operation.*—No brain tumor found; on removal of the roof of the right orbit definite osteoma of the roof found

Case 21.—Woman, 26 years old, admitted on March 9, 1949

*History.*—Self-induced "abortion" attempted in a nonpregnant uterus six weeks ago; three weeks later, generalized headaches; recently, blurring of vision, dizziness and personality changes; patient left-handed

*Examinations.*—Neurologic: Ataxic gait and a slight central weakness on the left; patient semistuporous, irritable, disoriented and uncooperative

Ophthalmologic: Bilateral papilledema with hemorrhages in the right eye

Roentgenologic: Skull, essentially normal, with the pineal body centrally placed and perhaps pushed backward and downward

*Vestibular Tests.*—Diagnosis based on the perverted responses from both canals of the right ear and the vertical canal of the left ear; high sensitivity; good hearing: "suspected supratentorial lesion, near the midline" (March 10, 1949)

*Ventriculogram.*—Done five days later; changes suggestive of a large lesion mass on the right frontal lobe

*Operation.*—Abscess in the right frontal lobe (Dr. Schuerman)

*Microscopic Diagnosis.*—Well encapsulated brain abscess

*Autopsy.*—A cavity, 5 by 3 cm., found on the anterior portion of the right frontal lobe; lining ragged and reddish brown; brain tissue surrounding the cavity hemorrhagic and soft

Case 22.—Man, 52 years old, admitted on Feb. 16, 1949

*History.*—Patient in the Graduate Hospital three months ago when right frontal abscess removed from the right frontal lobe; urinary incontinency, generalized fatigue and headaches one month ago; recently, mental obtundity and weakness on the right side

*Examinations.*—Neurologic: Central weakness on the right side of the face; weakness of the right arm; pronounced mental obtundity

Ophthalmologic: Early bilateral papilledema; small hemorrhages in right eye

*Vestibular Tests.*—Diagnosis based on perverted responses from the horizontal canal of the left ear; extreme sensitivity; generally good hearing: "suspected supratentorial lesion, left side"

*Ventriculogram.*—Done after the vestibular examination; lesion mass in the left frontal lobe

*Operation.*—Brain abscess in the left frontal lobe (Dr. Schuerman)

*Microscopic Diagnosis.*—Long-standing, well encapsulated brain abscess; mostly Staphylococcus aureus on culture of material from abscess

*Autopsy.*—Death three months afterward; "chronic abscess in the left frontal lobe; chronic leptomeningitis; ependymitis"

CASE 23.—Woman, 68 years old, admitted on March 13, 1949

*History.*—Personality changes and mental confusion three months before admission; occipital headaches and vomiting three weeks ago; recently, very sleepy

*Examinations.*—Neurologic: Almost complete mental block; disorientation, confusion and difficulty in speech; neck rigid; all reflexes depressed; positive Hoffmann sign on the left; positive Babinsky sign; patient poorly nourished, dehydrated and in advanced senility; incontinence of the bowels and bladder

Ophthalmologic: Bilateral papilledema, apparently of long standing

Roentgenologic: Skull, essentially negative; chest, diffuse nodular increased opacity in both pulmonary fields; appearance of lesion, hematogenous tuberculosis or generalized carcinomatosis

*Vestibular Tests.*—Diagnosis based on the perverted responses from both horizontal canals: "supratentorial lesion, near the midline", suspected despite slight sensitivity to the test; hearing very difficult to evaluate due to poor cooperation; slightness of sensitivity to the test probably due to patient's general depression, confusion and extreme uncooperation

*Ventriculogram.*—Done two days later; questionable evidence of tumor of the third ventricle

*Operation.*—Tumor in the third ventricle, probably metastatic (Dr. Schuerman)

*Microscopic Diagnosis.*—Malignant, rather anaplastic tissue

*Autopsy.*—Death one month later; autopsy not made because of refusal of family

PROTOCOLS OF CASES OF CEREBELLOPONTILE ANGLE LESIONS

CASE 24.—Woman, 59 years old, admitted on April 28, 1949

*History.*—Hearing loss in the left ear, tinnitus, dizziness; lately, diplopia; symptoms of eight years' duration

*Examinations.*—Neurologic: Dyssynergia in the left hand and arm; positive Romberg sign (falling to the left); ataxic gait with veering to the left; deafness in the left ear

Ophthalmologic: "On the basis of the Bárány findings, not at all consistent." No papilledema; corneal reflexes intact; "arteriosclerotic retinopathy, only pathologic change found, sufficient to suggest possible cranial or cerebral arteriosclerosis"

*Vestibular Tests.*—Diagnosis based on the spontaneous past pointing with the left hand; tendency to fall to the left; poor resistance to the pelvic girdle test;

no cochlear or vestibular responses from the left ear after five minutes of douching; no sensitivity: "suspected cerebellopontile angle lesion on the left side" (April 29, 1949)

Roentgenologic: Report on the next day: "moderate degree of destruction of the inner half of the petrosa on the left due to cerebellopontile angle tumor"

*Operation.*—Left cerebellopontile angle tumor (Dr. Groff)

*Microscopic Diagnosis.*—Perineural fibroblastoma

CASE 25.—Woman, 40 years old, admitted on May 27, 1949

*History.*—Staggering to the right, occipital headaches, vertigo, diplopia, nausea and vomiting, of four months' duration

*Examinations.*—Neurologic: Dyssynergia on the left side of the body; dysdiadokokinesis; hyperreflexia; positive Hoffmann sign on the left; spontaneous nystagmus

Ophthalmologic: Bilateral papilledema

Roentgenologic: Skull, marked destruction of the posterior clinoid process and dorsum sellae indicative of increased intracranial pressure; otherwise, normal

*Vestibular Tests.*—Diagnosis based on spontaneous nystagmus; postural nystagmus (apparent change of direction with change of position); spontaneous vertigo; spontaneous past pointing with both hands to the left; tendency to fall to the right, regardless of which direction head was turned; poor resistance to the pelvic girdle test; perverted and inverted responses from the vertical canal of the right ear; perverted responses from the horizontal canal of the right and left ear; no sensitivity; grossly good hearing: "suspected lesion of the posterior cranial fossa, inclined to the right"

*Operation.*—Suboccipital craniectomy (Dr. Schuerman); cerebellopontile angle tumor on the right side; lesion not localized in the angle because vestibular tests findings used as diagnostic base; audiogram not made; hearing tested only with tuning forks; believed that patient could hear from both ears; ears were not masked during tuning fork examination; may account for the impression "grossly good hearing for high tones; some loss for low tones"

*Microscopic Diagnosis.*—Microscopic report in this case not obtainable (June 15, 1949)

CASE 26.—Man, 46 years old, admitted Jan. 17, 1949

*History.*—Progressive deafness in the left ear of two years' duration; clumsiness on the left side of the body and staggering gait with tendency to fall to the left noted two months before admission; lately, paresthesias, numbness of the left side of the face, nausea and blurring of vision

*Examinations.*—Neurologic: Spontaneous nystagmus; slightly decreased left corneal reflex; paresthesia in the third division of the fifth cranial nerve; slight dysmetria; adiadokokinesis of the left leg and hand; slightly ataxic gait; deafness in the left ear; questionable involvement of the ninth cranial nerve

Ophthalmologic: Negative results except for the spontaneous nystagmus

Roentgenologic: Skull, essentially normal

*Vestibular Tests.*—Diagnosis based on the horizontal and vertical spontaneous nystagmus; poor cochlear and vestibular responses from the left ear; oblique nystagmus elicited from the horizontal canal of the left ear; perverted responses from both canals of the right ear; good hearing in the right ear; no sensitivity;

very poor hearing in the left ear: "suspected lesion of the posterior cranial fossa near the left cerebellar lobe"

*Operation.*—Tumor deeply situated on the left cerebellopontile angle (Dr. Groff)

*Microscopic Diagnosis.*—Neurinoma, periacustic, strongly fibroblastic

CASE 27.—Man, 48 years old, admitted on June 8, 1949

*History.*—Loss of hearing in left ear, tinnitus, headaches and some unsteadiness of the left leg and arm; lately, generalized weakness and occasional paresthesia of the left side of the face

*Examinations.*—Neurologic: Spontaneous nystagmus; positive Romberg sign; dysdiadokokinesis in left arm; dyssynergia in left arm; corneal analgesia in left eye; veering to the left on walking

Ophthalmologic: Spontaneous nystagmus; disks oval and flat with slight haziness of the nasal margins on fundus examination; no hemorrhages or exudates; corneal anesthesia not found; no papilledema; arteriosclerotic retinopathy, grade 2; "cerebellopontile angle tumor suspected in spite of absence of corneal anesthesia, and papilledema and presence of other findings" (June 10, 1949)

*Vestibular Tests.*—Done two days before ophthalmologic examination; diagnosis based on the spontaneous nystagmus; positional nystagmus which changed its direction when positions changed; tendency to fall to the left; poor resistance to the pelvic girdle test; spontaneous past pointing with both hands; no responses from both canals of the left ear after being douched for more than three minutes; perverted responses from both canals of the right ear; deafness in the left ear; no sensitivity: "cerebellopontile angle tumor, left side"

*Operation.*—Tumor in the left cerebellopontile angle (Dr. Schuerman)

*Microscopic Diagnosis.*—Microscopic report in this case not obtainable (June 29, 1949)

CASE 28.—Woman, 35 years old, admitted on March 21, 1949

*History.*—Deafness in right ear, tinnitus, dizziness, feeling of heaviness on the right side of the face, of three years' duration; on admission, staggering gait and loss of facility in use of the right hand

*Examinations.*—Neurologic: Depressed corneal reflexes on right side; deafness in the right ear; ataxic gait; increased reflexes; spontaneous nystagmus

Ophthalmologic: Bilateral papilledema; small hemorrhage in the left eye; spontaneous nystagmus

*Vestibular Tests.*—Diagnosis based on the horizontal and vertical spontaneous nystagmus; lack of vestibular and cochlear responses from the right ear; perverted responses from both canals of the left ear; no sensitivity: "cerebellopontile angle tumor on the right side"

*Operation.*—Tumor in the right cerebellopontile angle (Dr. Groff)

*Microscopic Diagnosis.*—Perineural fibroblastoma of the eighth cranial nerve

CASE 29.—Man, 63 years old, admitted on June 9, 1949

*History.*—Deafness in the right ear of twenty years' duration; tinnitus, numbness of the right side of face, tearing from the right eye, salivary dripping from the right side of the mouth, loss of taste from the right side of the tongue, of three months' duration; lately, intermittent headaches, unsteadiness in gait and some loss of hearing in the left ear

*Examinations.*—Neurologic: Peripheral paralysis of the fifth, seventh, and eighth cranial nerves on the right; dysdiadokokinesia on the right arm; absence of abdominal reflexes on the right; ankle clonus on the right; possible increase in the reflexes on the right; corneal analgesia of the right eye; spontaneous nystagmus

Ophthalmologic: Spontaneous nystagmus; disks oval and flat; normal color; clear outline; no hemorrhages or exudates; macula and periphery clear; no papilledema; slight diminution of sensitivity of the cornea of the right eye

*Vestibular Tests.*—Diagnosis based on the rotatory and horizontal spontaneous nystagmus; positional nystagmus; spontaneous pastpointing with both hands to the right; poor pelvic girdle movements; tendency to fall to the right and backward; no cochlear or vestibular responses from the right ear; perverted and inverted responses from the vertical canal of the left ear; perverted responses from the horizontal canal of the left ear; pronounced mixed type of deafness in the left ear; no sensitivity: "strongly suspected cerebellopontile angle lesion on the right side"

*Operation.*—Tumor in the right cerebellopontile angle (Dr. Schuerman)

*Microscopic Diagnosis.*—Neurinoma of the acoustic nerve

Case 30.—Woman, 42 years old, admitted on April 19, 1949

*History.*—Deafness in the right ear of one year's duration following an injection of procaine hydrochloride by a dentist; involvement of the trigeminal and auditory nerves on the right side of the face; lately, unsteadiness in gait; blurring of vision; numbness of right side of face

*Examinations.*—Neurologic: Paralysis of the facial nerve on the right; deafness in the right ear; decreased motion of the soft palate on the right side; complete anesthesia of the right side of the face, including the cornea; staggering gait; positive Romberg sign; dyssynergia bilaterally, more evident on the right; mind clear and alert

Ophthalmologic: No papilledema; corneal anesthesia on the right

Roentgenologic: Essentially negative results

*Vestibular Tests.*—Diagnosis based on the spontaneous nystagmus; poor resistance to the pelvic girdle test; the elicitation of one or two twitches of the eyes after douche of the right ear for only three minutes, indicating practically dead labyrinth; perverted responses from the horizontal canal of the left ear; very slight sensitivity; pronounced deafness in the right ear: "strongly suspected central lesion; possibility of cerebellopontile angle lesion on the right side"

*Operation.*—Tumor on the right cerebellopontile angle (Dr. Groff)

*Microscopic Diagnosis.*—Perineural fibroblastoma of the eighth cranial nerve

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# HISTOLOGIC PICTURE OF INHERITED NERVE DEAFNESS IN MAN AND ANIMALS

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HEREDITARY factors play an important role in the cause of nerve deafness. A thorough knowledge of their mode of action and their clinical and anatomic manifestations is essential for the proper recognition, management and prevention of many forms of hearing loss. The question of prevention is of particular importance, since hereditary deafness has not been and is not likely to be amenable to any kind of therapy.

In the last twenty years knowledge in this field has greatly increased, particularly because of the extensive research done by animal geneticists. The published material is, however, scattered throughout a large number of journals, monographs and dissertations, some of which are hardly accessible for the average reader. Only very few of the reports have appeared in the American otolaryngologic literature, and a comprehensive up-to-date review of the findings is entirely missing.

In this paper it has been attempted not only to report the changes in 2 cases of what appears to be inherited nerve deafness in man but also to give a comprehensive review, in the hope of facilitating future research in this way.

A proper grouping of the anatomic findings in hereditary nerve deafness is very difficult, and none of the classifications thus far given is completely satisfactory.

In the past, a distinction has been made frequently between congenital and acquired forms of deafness. This distinction is not exact, however, because deafness present at birth is not necessarily inherited, in the biologic sense of the word. The deafness might have been acquired during intrauterine life; for instance, as the result of a rubella infection of the mother during the first months of pregnancy. On the other hand, inherited deafness is not always noticeable at birth but might become manifest long afterward.

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Steurer, in a widely accepted classification, distinguished between two types of deafness on the basis of the anatomic findings: one with regressive changes in the end organ, the cochlear nerve and its pathways and the other with evident developmental anomalies in the cochlea. The regressive changes develop either "spontaneously" without any apparent extrinsic cause or secondary to known external factors, such as inflammations, traumas and hemorrhages. "Spontaneous" regressive changes and outright developmental anomalies form the anatomic basis of inherited deafness, secondary regressive changes the basis of acquired deafness.

In many instances a clearcut distinction between these types is much easier in theory than in practice, especially when the findings are limited to the membranous parts of the cochlea.

It is self evident that changes found in the temporal bone of a deaf-mute should never be considered as congenital malformations solely on the basis of a history of deafness since birth or deafness in the family. They might be caused by a disease of the inner ear which started during intrauterine or early postnatal life. The function of the inner ear might have been destroyed before it had been possible to decide with certainty if hearing had previously been present. Before being interpreted as congenital malformations, findings should be compared with the pictures of known regressive changes. The diagnosis of congenital malformation should be made only if the anatomic picture in question is different from all known pictures of the different stages of spontaneous or secondary regressive changes of the inner ear. Occasionally, in doubtful cases, the presence or absence of other malformations in the ear or other parts of the body may, but must not necessarily, be a diagnostic aid. Sometimes, for instance, acquired deafness has been found together with a malformation of the endolymphatic duct and sac (Alexander, 1907).

Of great importance for the proper evaluation of the findings are the results of experimental studies on animals carried out by Herzog, Wittmaack, Steurer, Runge, Werner and others. They show clearly that changes frequently present in the cochlea of deaf-mutes, previously regarded as developmental anomalies, could be reproduced through the action of certain chemicals, such as calcium chloride, pilocarpine hydrochloride, guanidine and arsenic acid. Among others, the following changes could be reproduced: ectasia or collapse of Reissner's membrane; a turning over, separation or adherence to surrounding formations of the lamina tectoria; formation of a nuclei-containing sheath around the lamina tectoria; complete disappearance of Corti's organ or its transformation into a low mound consisting of flat or more cuboidal cells; development of formations resembling epithelial proliferations in the stria vascularis, or the development of thin strings

between different parts of the cochlear duct. Changes might also be found in the maculas of the saccule and utricle, but not as extensive as those in the cochlea. In addition, one might find an atrophy of the cochlear fibers and of the spiral ganglionic cells caused, in Wittmaack's opinion, by a direct toxic effect of the substance producing the labyrinthine changes. Such changes are, according to Wittmaack, the result of increased production of endolymph, of a labyrinthine hydrops, which causes irreparable damage to the labyrinthine structures. Mild degrees of hydrops lead to the changes just described, a so-called posthydropic neuroepithelial degeneration; severe degrees lead to a posthydropic necrosis of the soft parts of the labyrinth. Wittmaack's assumption, that the changes are the result of a simple quantitative increase in the endolymph production and are noninflammatory, has become untenable for many reasons (Werner). There is a gradual transition between the changes just described and the obviously inflammatory changes of the labyrinth. The same substances, depending on their concentration in the labyrinthine fluids, are able to produce either of these changes.

In view of these facts, many cases which were previously believed, without exception, to belong to the group of developmental anomalies must now be explained as regressive changes.

The histologic differentiation of spontaneous and secondary regressive changes has remained very difficult if not impossible in many instances, particularly in those cases in which they are superimposed on developmental anomalies.

## REVIEW OF THE CASES WITH REGRESSIVE CHANGES IN THE END ORGAN

1. *The Findings in Animals.*—Analysis of the anatomic findings in man is aided by the fact that in certain animals, such as cats, dogs, guinea pigs and mice, inherited forms of deafness are known to exist. By breeding strains of such deaf animals, it is possible to make a thorough study of the hereditary pattern and a histologic examination of sufficient numbers of all age groups to clarify the pathogenesis of the aural changes.

The most extensive studies on this subject were carried out by geneticists on mice and other rodents. (For details see Grüneberg, 1943 and 1947, and Nachtsheim.) In mice, the eleven, independent, recessive genes for the following types have thus far been described, each of which causes deafness associated with locomotor disturbances. Additional genes are likely to become known in the future.

1. Waltzing mouse (Rawitz, Baginsky, Alexander and Kreidl, Quix, van Lennep, Wittmaack, Tatsumi, Gates, Hattori, von Gilse, Yasuhara and others)

2. Shaker 1 (Lord and Gates, Meng, Grüneberg, Hallpike and Ledoux)
3. Shaker 2 (Dobrovolskaia-Zavadskaia)
4. Jerker (Grüneberg, Burnett and Snell)
5. Kreisler (Hertwig)
6. Pirouetting mice (Woolley and Dickie)
7. Dervish (Woolley, cited by Grüneberg, 1947)
8. Shaker with syndactyly (Hertwig)
9. Fidget (Grüneberg, 1943)
10. Shaker short (Dunn, Bonnevie)
11. Varitint waddler (Cloudman and Bunker)

The semidominant genes for kinky tail and syndactyly in the mouse are also often associated with deafness, but the data so far published are still scanty (Dunn and Caspari). Circlers described by Laanes and MacDowell show hypersensitivity to loud sounds during early age. This hypersensitivity diminishes in later life.

Mutations of a similar type have been found in every other species of laboratory rodents. Waltzing and deafness have been observed in the American mouse of the genus Peromyscus (Dice, Watson). Among the subspecies of the deermouse (Peromyscus maniculatus), P. maniculatus bairdii is never deaf; in P. maniculatus artemisiae at least some of the young mice can hear for several months, but all the old ones are deaf. The same conditions prevail in the cactus mouse, P. eremicus (Dice). Waltzing without deafness has been observed in the rat (King); waltzing and deafness, in the guinea pig (Ibsen and Risty, Ibsen, Lurie, Lurie and Dempsey), and waltzing with undetermined sense of hearing, in the rabbit (Cole and Steele, Cogan).

While all the other members of the waltzer-shaker group become deaf early in life, fidgets do not. Young fidgets about 3 weeks of age are actually hypersensitive to sounds; later, the hearing decreases, and from the age of 3 to 4 months onward, fidgets show no definite auditory reaction.

Histologic examinations have been made of comparatively few of these mutations. Changes were observed to be confined mostly to the cochlea, but sometimes the macula sacculi is also affected; waltzing and shaking seems to be due to a lesion of the central nervous system and not to changes in the vestibular end organ (Kuiper, Cunge, Zimmermann, Lurie and Dempsey).

The details of the pathologic changes in waltzing mice are still incompletely known, in spite of considerable literature on this subject. It is generally agreed, however, that the labyrinth of waltzers is com-

pletely normal at birth. According to van Lennep, the cochlea shows no morphologic abnormalities up to five days after birth, although at this stage changes in the vessels of the stria vascularis can be recognized. Ten days after birth, changes in the hair cells of the organ of Corti become visible and increase progressively thereafter with age. Later still, a degeneration of the spiral ganglion becomes noticeable.

In waltzing guinea pigs, the degeneration of Corti's organ begins at birth or near the termination of intrauterine life. It starts in the first and second turn and progresses toward the helicotrema. It is not a gradually progressive degeneration but an irregular one. A small remnant near the helicotrema may remain normal. The organ is finally replaced by a simple cuboidal epithelium. The external sulcus cells also degenerate. The stria vascularis appears either atrophic or normal in different parts of the same cochlea. No accurate estimate could be made of the relation of the changes found in the stria vascularis to those found in the organ of Corti. The cochlear nerve fibers and the cells of the spiral ganglion degenerate at a much slower rate. The macula sacculi remains normal.

The Shaker 1 mutation resembles the waltzing mouse and guinea pig in many ways. Starting when this mutation is 22 to 30 days old, deafness develops rapidly within a few days.

Histologic changes are again confined to the cochlea and start twelve days after birth in a fully developed organ of Corti. They first appear together in Corti's organ and in the stria vascularis. In Corti's organ the degeneration gradually affects all the outer hair cells, then the inner hair cells. Eventually, the organ becomes reduced to a small compact hillock of undifferentiated cells. The stria vascularis shows a progressive loss of cytoplasm of the epithelial cells, with relative increase of the nuclei and the vascular spaces and subsequent reduction in the volume of the stria. The vessels themselves do not show definite changes. Reissner's and tectorial membranes remain unchanged. The change in the spiral ganglion starts when the mutation is aged 50 days, apparently, secondary to that in the peripheral fibers in Corti's organ, and involves a progressive loss of ganglionic tissue.

The absence of visible changes in the vascular supply of the internal ear, in particular of the stria vascularis, precludes the possibility that the degenerative processes described are primarily of vascular origin. The following explanation is, according to Grüneberg (1943), more probable: During embryonic and early postnatal life, the cells of Corti's organ in mice receive their nutrition from a large vessel, the vas spirale. This vessel later undergoes atrophy. From that time onward, Corti's organ depends for nutrition entirely on the endolymph which is secreted from the stria vascularis. As long as the function of the vas spirale lasts, the structure of Corti's organ is perfectly normal. Degenerative changes

set in, however, after the vas spirale has disappeared, probably because the stria vascularis, which is visibly abnormal, fails to fulfil its secretory function properly.

Shaker 1 is, as mentioned, fully recessive. Heterozygotes are completely normal in their behavior and retain the sense of hearing throughout life. The same applies to animals heterozygous for the gene of waltzing. Cross breeding of the two genes gives an offspring which does not shake or run in circles but which has a tendency to become deaf from the age of about 3 months onward. There are, however, no histologic examinations of the ears of such animals on record.

In the three previously mentioned mutations the changes consist in a degenerative atrophy of the organ of Corti and of the stria vascularis, with secondary changes in the spiral ganglion. The vestibular part of the labyrinth remains unaffected. The exact relation between the changes in the stria and the organ of Corti is undetermined. Although there is no convincing evidence that the former invariably and necessarily precedes the latter, the assumption of Grüneberg that anomalies in the secretory activity of the stria are the main and primary cause seems not improbable.

The changes in the shaker short and the Kreisler mutations are entirely different from the findings just described and will be mentioned only for the sake of completeness. In both mutations, there is an anomalous development of the otocyst, in Shaker shorts due to an anomaly of the brain, in Kreislers due to an invagination of the otocyst at a greater distance from the neural tube than in normal embryos.

The characteristic association of locomotor and acoustic disturbances in rodents has no close parallel in other animals, such as deaf cats and dogs, or in man.

> On this view, the true counterparts on inherited deaf-mutism in man remain to be discovered in rodents. However, this does not detract from the medical interest which attaches to this material, for it enables us to gain information about labyrinthine physiology and pathology which could not be obtained in man, and which will ultimately find application in the understanding of human conditions [Grüneberg, 1947].

Recessive deafness has long been known to occur in cats and dogs with partial loss of pigmentation. It seems to be a general rule that animals with only a partial loss of pigmentation, no matter whether they possess white hair or are dappled, often show defects in the nervous system or the sense organs.

Among cats, deafness is prevalent in those with white coats and blue eyes. Some early observers regarded white cats as albinos or incomplete albinos, but it is now definitely established that the white of ordinary yellow-eyed or blue-eyed cats is dominant white and in no way related to albinism. Not all blue-eyed cats, however, are deaf. On the

other hand, deafness in white cats is not invariably accompanied by complete absence of yellow pigment of the eye. True albinos with white coat and pink eyes hear well (Bamber).

Genetic analysis of the findings in dogs is complicated, and knowledge of the subject is still incomplete.

Collies, Shetland sheep dogs, Old English sheep dogs, Norwegian dunker hounds, great Danes, dachshunds and fox hounds are often found in the pattern of color known among the dog breeders as merle. (See Wright, Wriedt, 1919, Anker and others.) In this pattern the pigment of the hair is greatly diluted in most areas but of full intensity in patches scattered irregularly through the coat. According to Mitchell, the merle pattern is caused by a single dominant gene for dilution.

Merle dogs show anomalies of the eyes and ears. Homozygous merle dogs are invariably deaf, and the deafness is almost always complete; heterozygotes are quite frequently deaf, but the deafness may be incomplete. The deafness seems to be due to a recessive factor which is linked with the merle factor (assumed for fox hounds by Philipps and Knight).

In dogs in which merle occurs it is often associated with ordinary recessive spotting. There is a very definite linkage between this spotting, anomalies of the eyes and deafness. The breeds in which this is best shown are white collies and bull terriers (Philipps and Knight).

Eye anomalies and deafness, either associated with or independent of each other, are also very common defects in Dalmatian or coach dogs. The genetic mechanism, however, is different. The Dalmatian spotting appears to be recessive to solid color (Philipps and Knight).

Histologic examination in animals shows changes which in most instances affect the entire pars inferior, cochlear duct and saccule, whereas the utricle and the semicircular canals are free from changes (Alexander, Beyer, Alexander and Tandler, Lagally, Fraser, van Gilse, Howe and Guild, Howe, Albrecht, Wolff, Ojala, Lurie and others). The findings in dogs and cats are sometimes identical with those in rodents; frequently, however, one finds either the characteristic changes of "posthydropic neuroepithelial degeneration" alone or in combination with those of degenerative atrophy. Degenerative atrophic changes in the cells of the spiral ganglion are usually noted, but they are often of much less intensity than those in the end organ.

All the previously mentioned instances of inherited deafness in animals show a strictly recessive hereditary trait. In man, also, the great majority of cases of inherited deafness show this trait, so-called sporadic deaf-mutism (Albrecht, Metzkes, Bigler, Sandel, Hanhart, Hughson and associates, Odiorne, Christoph, Hopkins, Macklin and associates and others).

2. *The Findings in Man.*—The anatomic findings in man are essentially identical with those in cats and dogs. Thus, the changes vary in their extent. In some instances they are limited to the stria and the organ of Corti, in others they involve the spiral ganglion and the cochlear nerve fibers also; in still others the primary acoustic centers in the brain stem and the central cochlear pathways in addition are involved. The primary acoustic nuclei have been normal in 3 cases (Albrecht, 1939, case 1, 2, 1940) and markedly atrophic in 2 cases (Quix and Brower, Nager, 1927, case 2). Again, the changes are always confined to the pars inferior and never involve the utricle and the membranous semicircular canals, as they do in many cases of proved acquired deafness.

Although the changes in the stria vascularis and in the organ of Corti are not necessarily strictly parallel, it is, nevertheless, most probable that the latter are secondary to changes in the stria (Scheibe, Schwabach, Quix, Denker, Wittmaack, Steurer and others). One must keep in mind, furthermore, that functional changes in the stria do not always manifest themselves in pronounced morphologic changes, particularly in the beginning stages. This can be concluded, for instance, from the findings in labyrinthine hydrops (true Ménière's disease) in man in which, in spite of unquestionable disturbances in the endolymph production, the stria has been apparently normal or at least free from obvious changes in all the cases thus far examined (Altmann and Fowler). If Grüneberg's assumption is correct that during fetal life the organ of Corti receives its nutrition not from the stria but from the vas spirale, and if this condition occurs not only in mice but also in other mammals, then changes present at birth would have to be attributed to anomalies in the vas spirale and not in the stria. It seems possible that if even a part of the function of the stria only is defective or lacking, the epithelium of the cochlear duct might become damaged and in some places even destroyed (Ojala). The different effect of various chemicals on the cochlear epithelium in animal experiments makes it conceivable that, depending on which part of function is disturbed, the changes in the cochlear epitheliums will vary.

If this assumption is correct, there is no essential difference between the findings with or without "posthydropic" changes. Both are, in the last analysis, caused by inherited anomalies in the secretion or the composition of cochlear endolymph.

The fact that in cats and dogs and in man the changes are usually not confined to the cochlear duct but also affect the saccule, while the utricle and the membranous semicircular canals remain normal, could indicate a close functional association between the two. It is mostly explained on a phylogenetic basis and is borne out in a certain way by the anatomic findings. A true branch of the cochlear division of the

eighth nerve, the cochleosaccular nerve of Hardy, leads from the cochlear nerve in the region of the spiral ganglion, near its basal end, to the posterior portion of the macula sacculi. Not enough is known, however, about the place and mechanism of the endolymph production and resorption in the saccule to give a more precise explanation of the findings. The fact that the cochlear duct and the saccule are sometimes collapsed while the utricle and the canals show a normal lumen could, according to Bast, be due to the function of the utriculoendolymphatic valve, which seems to prevent a collapse of the pars superior. Bast concluded this, aside from other evidence, from the findings in 2 children with a ruptured saccule and collapse of the cochlear duct but with normal distention of the pars superior. According to Werner, however, a rupture of the saccular wall would lead only to an equalization of the saccular and perilymphatic pressure and not, as assumed by Bast, to a decrease in pressure within the pars inferior with closure of the utriculoendolymphatic valve. That rupture of the saccule must not necessarily lead to collapse of the entire pars inferior is seen also in a case of endolymphatic hydrops (true Ménière's disease) with rupture of the saccule, reported by Lindsay.

According to Secrétan, the valve is not always functioning. In some cases there is a fold on the opposite wall which seems to permit a current from the utricle toward the endolymphatic duct but not in the opposite direction. This would explain why in certain malformations the pars inferior of the labyrinth is markedly dilated but the utricle intact.

The changes in the cochlear nerve fibers and the cells of the spiral ganglion are usually regarded as secondary to those in the organ of Corti. The findings in rodents are certainly suggestive of such a mechanism, although the possibility of a direct action of the anomalous endolymph on the nerve fibers or a coordinated hypoplasia or degenerative atrophy should also be considered. Not enough is known, however, about the functional relation between the hair cells and the cochlear nerve fibers and the cells in the spiral ganglion to permit definite conclusions.

The findings in the auditory nuclei and the central pathways could not possibly be explained as secondary to the cochlear changes; they probably represent either a primary hypoplasia or an inherited inferiority of these areas. They are unquestionably a coordinated anomaly.

If the cochleosaccular changes in certain forms of inherited deafness are actually the result of anomalies in the composition of the endolymph, it becomes understandable that they are morphologically indistinguishable from the changes in those forms of acquired deafness in which in early childhood a diffusion of toxic substances from the cerebrospinal fluid or from the middle ear, particularly through the round window, has taken place. A thorough investigation and genetic evaluation of the family history might be the only way for the proper differentiation of the cases in question.

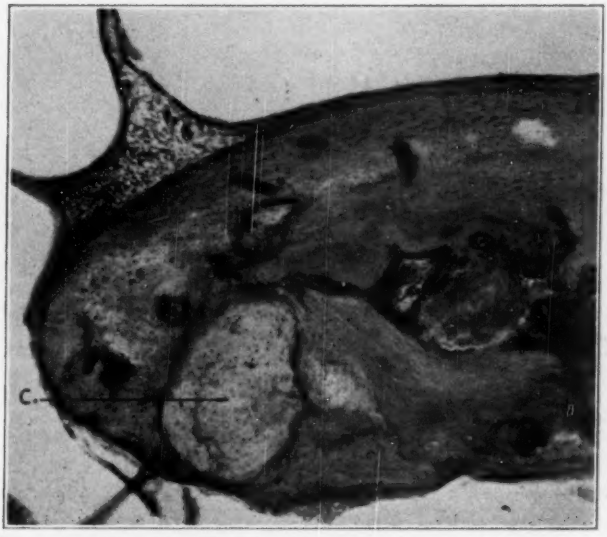


Fig. 1 (case 1).—Right side. Rounded mass of cartilage (*c*) in the short process of the incus.

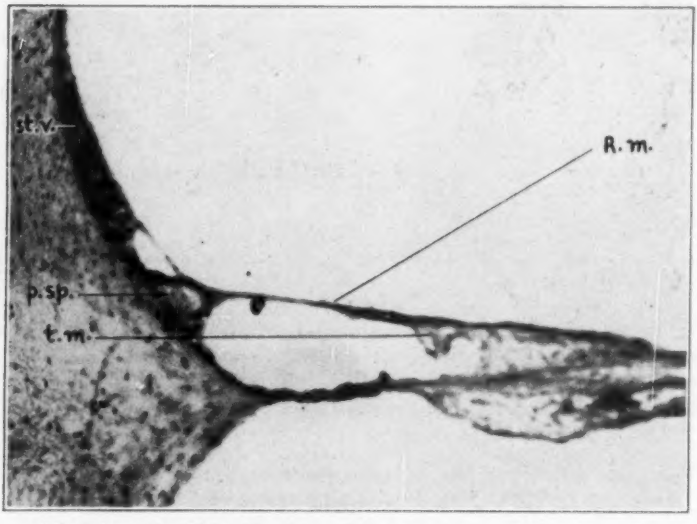


Fig. 2 (case 1).—Right side. Lower half of basal turn. Reissner's membrane (*R. m.*) is collapsed and adherent to stria vascularis (*st. v.*) and prominentia spiralis (*p. sp.*). Corti's organ has disappeared; tectorial membrane (*t. m.*) is turned upward and adherent to Reissner's membrane.

REPORT OF TWO CASES

The following observations on 2 cases show quite well the great diagnostic difficulties encountered in these cases. Both come from the histologic collection of the Ear, Nose, and Throat Department of the University of Vienna Medical Faculty.

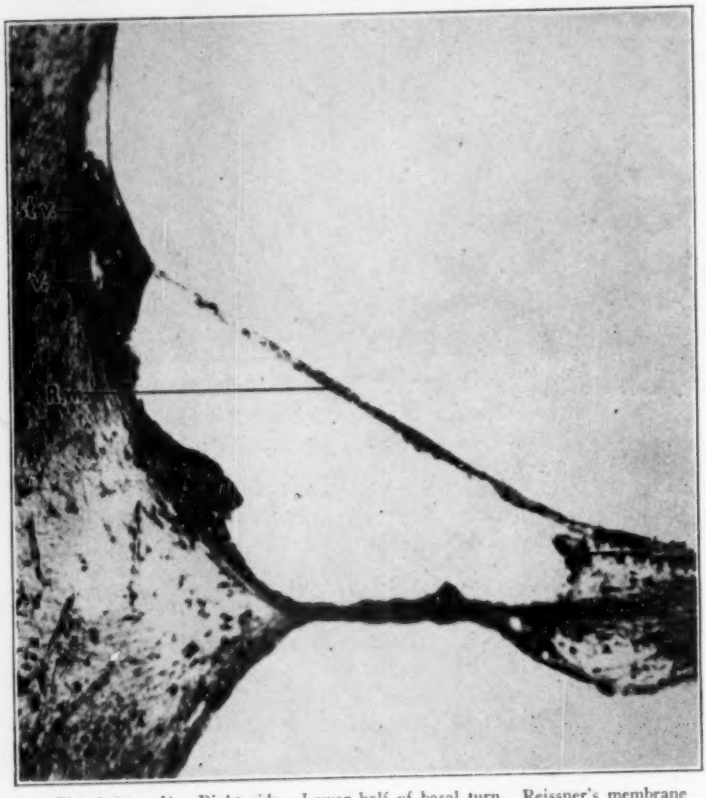

Fig. 3 (case 1).—Right side. Lower half of basal turn. Reissner's membrane (*R. m.*) is adherent to the stria vascularis (*st. v.*), which is represented by a circumscribed epithelial thickening containing a wide vascular space (*v.*). The organ of Corti has disappeared; tectorial membrane (*t. m.*) is tucked into the internal spiral sulcus.

Case 1.—J. B., a 22 year old woman, died of multiple sarcomas of the bones. Both parents and most of the blood relatives for the last three generations were deaf-mutes. The patient had no brothers or sisters. More detailed data on the family history are not available. The patient's intellectual development was normal, and up to her eighteenth year she was healthy except for the usual childhood diseases.

The histologic examination of the temporal bones (embedded in pyroxylin U. S. P., vertical sections, 20 microns thick, stained with hematoxylin and eosin) showed the following conditions.

Of the right ear, the external auditory meatus and drum membrane were normal.

The middle ear showed normal configuration and size. The mucosa was slightly hyperplastic. In the window niches, the attic, antrum and other recesses, a few thin mucosal folds or connective tissue bands were seen traversing the lumen. Sometimes they delimited small cystic spaces within the niches. In the oval window niche the tissue stood extended also to the crura of the stapes in the attic, to the head of the stapes and to the body and the short process of the incus. The inner

Fig. 4 (case 1).—Right side. Upper half of basal turn. Concrement in a striate vessel.

surface of the posterosuperior quadrant of the drum was covered with slightly hyperplastic mucosa. The petrous tip was mostly diploic. The perilabyrinthine cells were moderately well developed. The bulb of the jugular vein was high and the bony floor of the middle ear moderately thick. The pneumatization of the mastoid process was slightly impeded, and the mucosal lining of the cell showed the same hyperplastic changes as in the middle ear.

The contents of the middle ear were normal. The posterior end of the short process of the incus contained a small rounded mass of cartilage, less than 0.5 mm. in diameter, whose circumference reached the outer surface of the process. The

cartilage was sharply delimited in all parts, and nowhere could any evidence be noted of ossification progressing from the surrounding bone into the cartilage (fig. 1).

Of the inner ear, the osseous labyrinthine capsule was normal.

Of the cochlea, the modiolus, the spiral laminas and the interscalar septums were normal.

In the cochlear duct, Reissner's membrane had collapsed in the vestibular part. In the lower half of the basal turn the collapse was less marked. In one part, the membrane adhered to the prominentia spiralis (fig. 2) and in another part to the stria vascularis, which here showed a circumscribed thickening (fig. 3). In other parts thin epithelial strings extended from the prominentia spiralis to Reiss-

Fig. 5 (case 1).—Right side. Proximal half of the rugose portion of the endolymphatic duct. Pronounced hyalinization of the tunica propria extending into the folds (*f.*). Large area of calcification (*c.*).

ner's membrane or to the tectorial membrane which, itself, adhered to Reissner's membrane. From the upper half of the basal turn upward, the cochlear duct was of normal width.

The organ of Corti had completely disappeared in the vestibular part and in the lower half of the basal turn; its upper half showed a low mound of undifferentiated cells. In some parts of the lower middle turn, cells resembling pillar cells could be observed. In the upper turn there was again a low mound of undifferentiated cells.

The tectorial membrane was either swollen and tucked into the internal spiral sulcus (fig. 3), turned upward and adherent to Reissner's membrane (fig. 2), or turned backward and overlying the limbus spiralis (fig. 7). It is, as a rule, covered with a sheet of flat epithelial cells.

The stria vascularis was completely atrophic in the vestibular portion and in the lower third of the basal turn. It then started as circumscribed epithelial

thickening (fig. 3) and gained a normal appearance toward the upper half of the basal turn, retaining it through the middle and apical turn. In the basal parts of the thickened portion of the stria, a relatively wide empty vascular space was noted (fig. 3). In the upper half of the basal turn was seen some homogenous, rounded, pink-staining concrements in the lumen of some capillaries, and in one

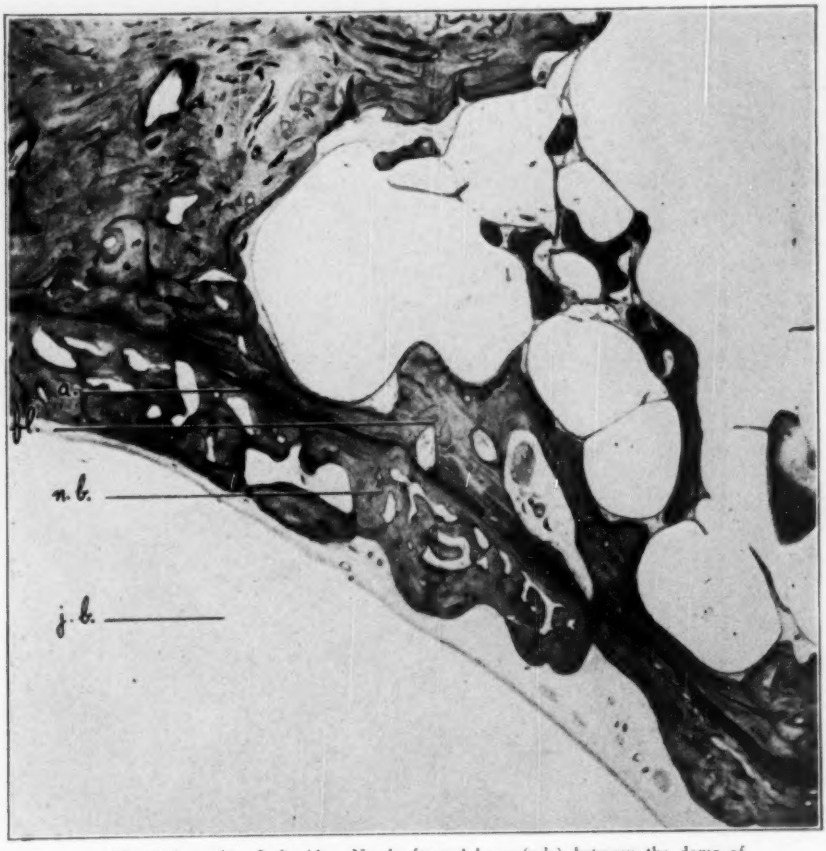


Fig. 6 (case 1).—Left side. Newly formed bone (*n.b.*) between the dome of the jugular bulb (*j. b.*) and the floor of the middle ear (*fl.*), separated from the floor by an apposition line (*a.*).

place was seen a deep blue-staining concrement in another capillary (fig. 4). A similar but smaller concrement was seen in the lower part of the middle turn.

The prominentia spiralis and the sulcus spiralis externus were covered with a lower epithelium in the vestibular part and in the lower half of the basal turn.

In the upper turns they appeared normal. The vas spirale was recognizable in most sections.

The crista and limbus laminae spiralis, the spiral ligament and the sulcus spiralis internus did not show definite pathologic changes.

The aquaeductus cochleae contained numerous "corpora amylacea."

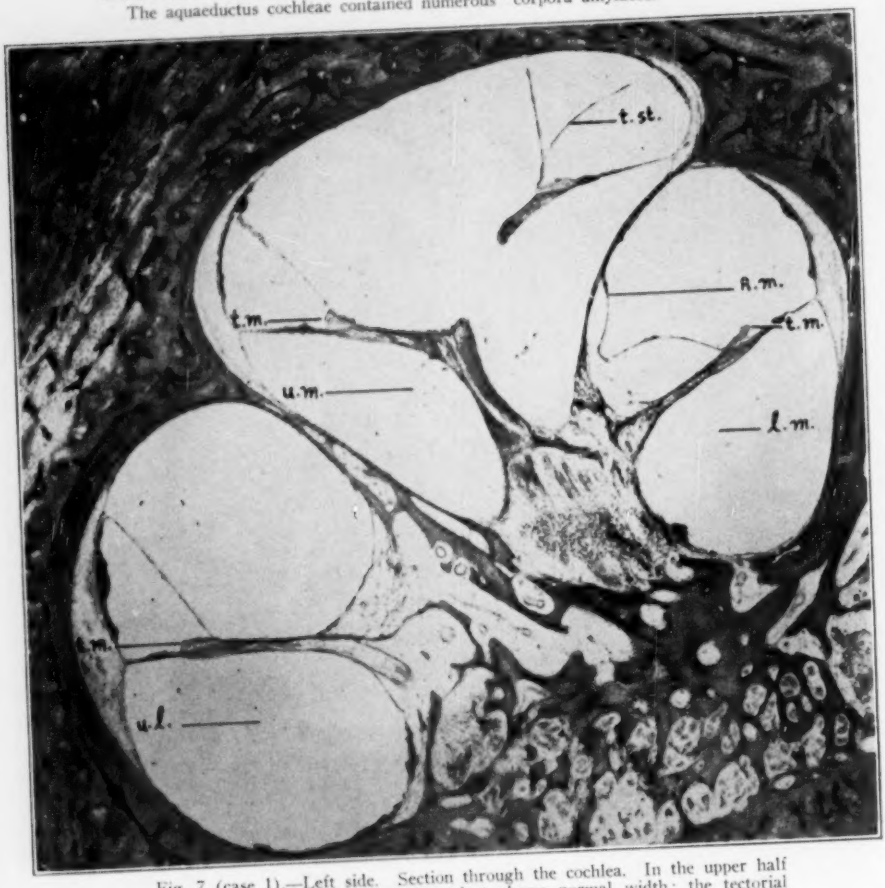


Fig. 7 (case 1).—Left side. Section through the cochlea. In the upper half of the lower turn (*u.l.*) the cochlear duct shows normal width: the tectorial membrane (*t. m.*) is tucked into the internal spiral sulcus. In the lower half of the middle turn (*l. m.*), Reissner's membrane is markedly ectatic, the tectorial membrane (*t. m.*) turned backward and overlying the crista spiralis. In the upper half of the middle turn (*u. m.*) the lumen shows normal width; the tectorial membrane (*t. m.*) is turned upward and adherent to Reissner's membrane (*R. m.*). In the upper turn the lumen is traversed by a thin tissue string (*t. st.*).

The spiral ganglion showed a slight reduction in the number of ganglionic cells in the vestibular part and in the lower basal turn with a correspondingly slight

reduction in the number of cochlear fibers. No abnormalities were noted, however, in the remaining cells and fibers in the relatively thick sections stained with hematoxylin and eosin.

The saccule had collapsed and the macula appeared atrophic. The saccular fibers seemed to be somewhat diminished in numbers.

The utricle and the membranous semicircular canals did not show definite pathologic changes.

In the proximal half of the rugose portion of the endolymphatic duct, the tunica propria showed a marked hyalinization, extending into the folds which project into the lumen. An area of calcification was seen extending from the endosteal

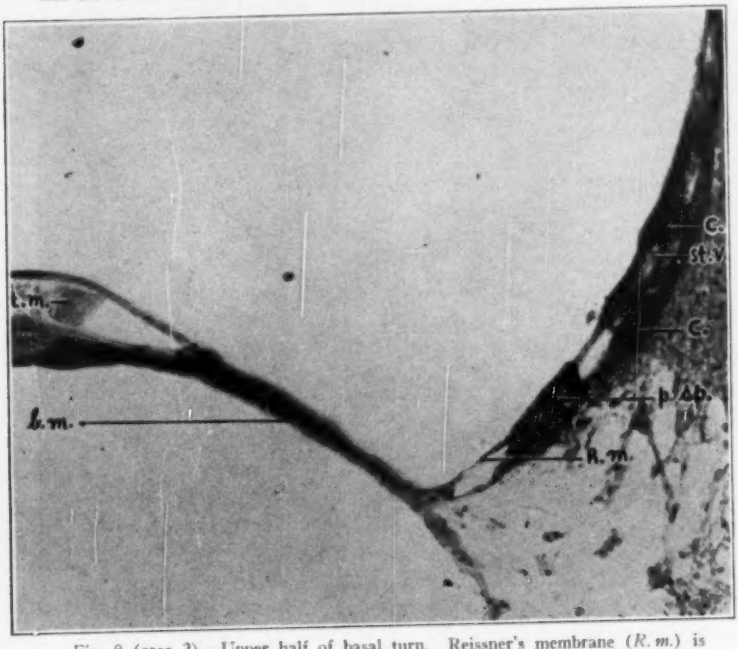


Fig. 8 (case 2).—Upper half of basal turn. Reissner's membrane (*R. m.*) is adherent to the stria vascularis (*st. v.*), prominentia spiralis (*p. sp.*) and the basilar membrane (*b. m.*). Corti's organ has disappeared; the swollen tectorial membrane is tucked into the internal spiral sulcus. The stria vascularis (*st. v.*) is atrophic and contains concrements (*c.*).

lining of the duct into the stroma. At the periphery of this area were fine granular deposits of calcium (fig. 5). Smaller areas of calcification were seen in some of the folds which project into the lumen. The peripheral part of the rugose portion and the sac proper appeared normal.

The contents of the internal auditory meatus appeared normal. No definite decrease in the number of cells of the vestibular ganglion could be recognized.

Of the left ear, the external auditory meatus and the drum membrane were normal.

The middle ear and the mastoid process showed approximately the same changes as those on the right. The jugular bulb was elevated. Between the dome of the bulb and the bony floor of the middle ear was interposed a 1 to 2 mm. thick layer of lamellar bone with large marrow spaces. This layer was separated from the bone of the floor proper by a well marked apposition line (fig. 6). The wall and the lumen of the jugular bulb itself seemed normal. At the posterior end of the short process of the incus was a small rounded mass of cartilage, measuring about 0.5 mm. in diameter.

Of the inner ear, the osseous labyrinthine capsule was normal.

The osseous framework of the cochlea was normal.

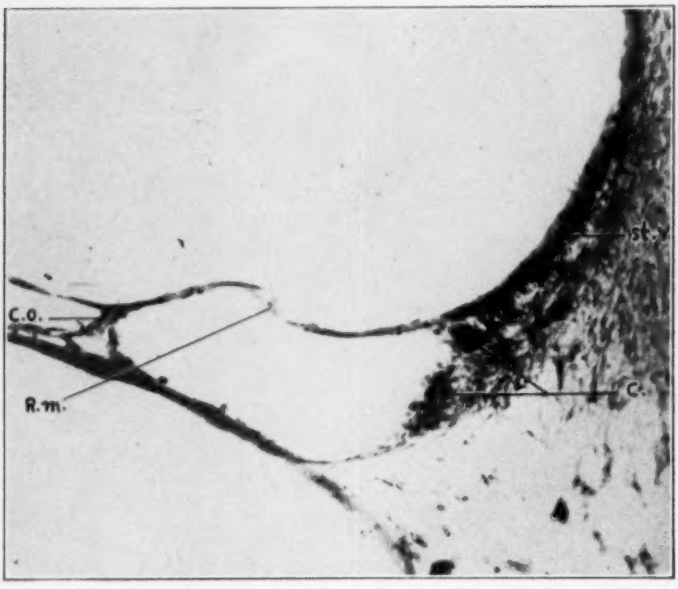


Fig. 9 (case 2).—Upper half of basal turn. Reissner's membrane (*R. m.*) is collapsed and touches the remnants of Corti's organ (*C. o.*) and the stria vascularis (*st. v.*). The prominentia spiralis contains concrements (*c.*).

In the cochlear duct, Reissner's membrane had collapsed in the vestibular end part and the lower half of the basal turn. It had come in contact with the atrophic stria, the limbus laminae spiralis, the prominentia spiralis in some places and also the remnants of the organ of Corti. In the upper half of the basal turn the membrane occupied a normal position; in the lower half of the middle turn it was markedly ectatic, and in the upper half and the apical turn it was again in a normal position (fig. 7). In two places, in the middle parts of the basal turn and in the apical turn, thin tissue strings traversed the lumen of the cochlear duct from the stria vascularis to the membrane of Reissner.

The organ of Corti was represented in all turns by a small mound of cells, between which no differentiation could be made.

The tectorial membrane showed the same changes as on the right side.

The stria vascularis was atrophic in the vestibular part of the cochlear duct and the lower half of the basal turn. In one part, toward the beginning of the upper half, a circumscribed thickening of the epithelium was noticed, the basal part containing a deep blue-staining, ovoid concrement. Another circumscribed thickening of the epithelium of the stria was found in the upper half of the basal turn and again contained a deep blue-stained concrement. In the other turns the stria looked normal.

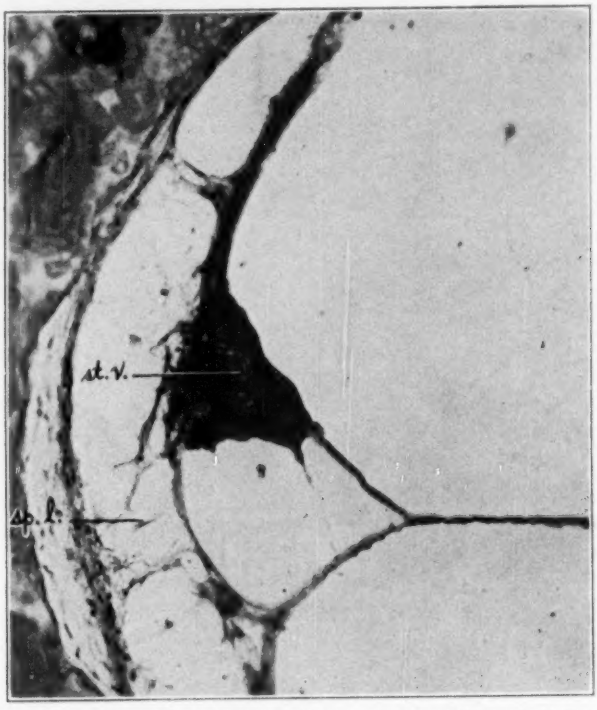


Fig. 10 (case 2).—Upper half of the middle turn. The stria vascularis is represented by a wartlike excrescence (*st. v.*). The stroma of the spiral ligament (*sp. l.*) is extremely rarefied.

The other parts of the cochlea and the saccule showed the same changes as those on the right.

The utricle and membranous semicircular canals and the endolymphatic duct and sac appeared normal.

Case 2.—J. J., a youth, died at the age of 20. Clinical data were not available.

The histologic examination of the temporal bones (embedded in pyroxylin, vertical sections 20 microns thick, stained with hematoxylin and eosin) showed almost identical findings on both sides.

The external auditory meatus and the drum membrane were normal.

The size and configuration of the middle ear were normal. The mucosa was thin, and there were hardly any mucosal folds or tissue strands in the various recesses and niches. The petrous tip was mostly diploic. The perilabyrinthine cells were moderately well developed, with the exception of the large and extensive infralabyrinthine cells. The dome of the jugular bulb was low; the contents of the middle ear were normal, and the antrum was of medium size. The mastoid bones showed normal pneumatization in the parts examined.

The osseous labyrinthine capsule of the inner ear was normal.

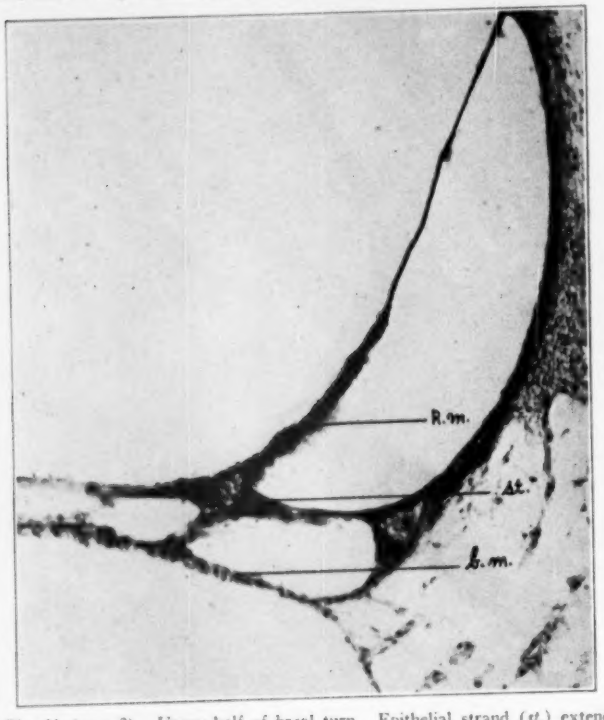


Fig. 11 (case 2).—Upper half of basal turn. Epithelial strand (*st.*) extending from the prominentia spiralis to Reissner's membrane (*R. m.*) and from there toward basilar membrane (*b. m.*).

The osseous framework of the cochlea showed normal configuration but was unusually thin.

In the cochlear duct, Reissner's membrane had collapsed in all turns from the vestibular to the apical end of the duct. The membrane had come into contact in many places with the stria vascularis, the prominentia spiralis and the limbus laminae spiralis. In many areas it touched the remnants of the organ of Corti or the basilar membrane itself (fig. 8); in others it approached the membrane without touching it.

The organ of Corti had either completely disappeared or was reduced to a flat mound of undifferentiated cells. Only in a circumscribed area in the upper half of the basal turn was it somewhat better preserved, and cells resembling pillar cells (fig. 9) or, in some parts, Deiters' or Hensen's cells could be made out. Homogenous pink-staining small globules were seen in certain parts between the degenerating remnants of Corti's organ.

The tectorial membrane was turned upward and adhered to Reissner's membrane at many points; it was also tucked into the internal spiral sulcus (fig. 8).

The crista and the limbus laminae spiralis showed a practically normal structure.

The stria vascularis was atrophic in the vestibular part and the entire basal turn. The tissue underneath the narrow band of epithelium contained numerous narrow capillary spaces. Some spaces were wider and contained ovoid homog-

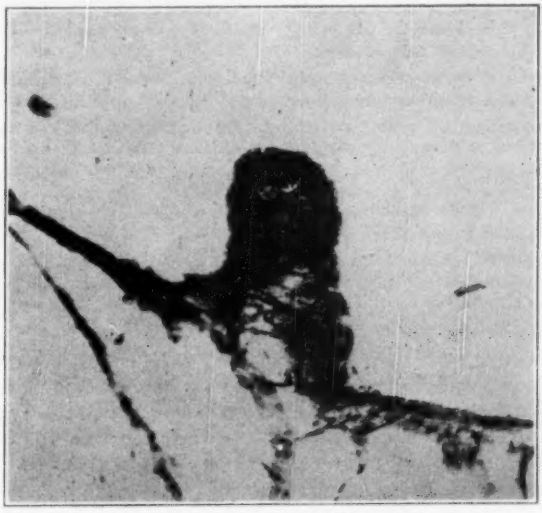

Fig. 12 (case 2).—So-called atypical epithelial formation in the medial wall of the sinus superior of the utricle.

enous blue and red-staining concretions; others were empty. Similar concrements were present also within some parts of the epithelium itself (fig. 8). In other places, the substriate tissue showed a looser texture, with formation of larger tissue spaces. In the upper turns the stria was markedly reduced in width and formed a wartlike excrescence bulging into the lumen of the cochlear duct (fig. 10). Here, again, concretions could be seen within the epithelium and within the capillary spaces; other capillary spaces were empty.

The prominentia spiralis and the sulcus spiralis externus were covered with flat epithelium. The vas spirale had collapsed in some sections; showed an empty lumen in others, and was invisible in still others. In some places the stroma contained concrements similar to those of the stria vascularis (fig. 9). In one place in the upper basal turn of the left side were strands of epithelial cells extending from the surface of the prominentia spiralis to Reissner's membrane and from

there down to the basilar membrane near the tip of the spiral ligament. A small hyaline concrement was contained within the epithelial strands. In other places an epithelial strand extended from the prominentia spiralis along the lower surface of Reissner's membrane in a medial direction toward the basilar membrane and the scanty remnants of Corti's organ (fig. 11).

The spiral ligament showed normal structure in the basal turn. In the upper turns the stroma was extremely rarefied (fig. 10). Only in the areas adjacent to the stria vascularis was the structure somewhat denser.

The spiral ganglion and the cochlear fibers looked normal in the upper turns. Toward the lower basal turn and the vestibular part was a moderate decrease in the number of the fibers and of the ganglionic cells. Definite degenerative changes were not recognizable in the sections.

The cochlear aqueduct was normal.

Collapse of the saccule was complete. The macular epithelium was atrophic; the otolithic membrane was not visible. The number of the saccular nerve fibers seemed only slightly reduced.

The utricle, the membranous semicircular canals and the endolymphatic duct and sac had a normal appearance.

In the medial wall of the sinus superior of the utricle near the opening of the common crus of the superior and posterior vertical canals, a low ridge was covered with columnar epithelium. On both sides of the ridge this epithelium gradually went over into the surrounding lower epithelium. The stroma of the ridge was rich in vessels but did not contain any nerve fibers. No trace of a cupula could be found on the ridge (fig. 12).

The nerves in the internal auditory meatus appeared normal in the sections, and no definite decrease in the number of the cells of the vestibular ganglion could be noticed.

### COMMENT

In case 1 was shown the characteristic changes of a "posthydropic neuroepithelial degeneration." There was a close parallel between the condition of the stria vascularis and the position of Reissner's membrane. The Reissner membrane was collapsed where the stria was atrophic and in a normal position or ectatic where the stria exhibited lesser or no morphologic changes. The concrements in several places in the basal parts of the stria either represented hyaline and calcified thrombi in some of the striate vessels or were the result of degenerative changes in the capillary endotheliums with calcification. They have been observed by other authors in similar cases (Nager, Wolff and others); without any other changes by Rollin, and by Yamakawa in a case of true Ménière's disease with labyrinthine hydrops. An unusual finding was the extensive hyalinization and calcification of the stroma in the proximal half of the rugose portion of the right endolymphatic duct. The only comparable observation has been reported by Schlander about a girl of 18 years in whom, in a similar location in both endolymphatic ducts, an exostosis was found extending from the osseous wall of the duct into the lumen. There were no other labyrinthine changes. Arnvig observed unilateral fibrosis of the perivascular tissue with formation of a

small fibroma. The hyalinization and calcification of the stroma could have been the preliminary stage of ossification and would eventually have led to formation of multiple exostoses. The changes were obviously unrelated to the changes in the inner ear.

The focus of cartilage in the posterior part of the short process of each incus was another unusual finding. It most probably represented a remnant of the original cartilaginous anlage which was excluded from its normal connection with the surrounding tissue during ossification and could therefore be regarded as a "choriston." Similar foci of cartilage have been found in the endochondral capsule of the labyrinth (Altmann, 1932).

The hyperplastic changes in the mucosa of the middle ear represented residua after low grade inflammatory infections of the middle ear in early childhood. The extensive apposition of bone to the lower surface of the floor of the left middle ear may have beep the result of a severer infection of the middle ear on this side with spread of the infection through the floor and subsequent reactive new formation of bone.

Reviewing the findings in the inner ear, one can say that again on the basis of the morphologic findings alone a decision between inherited or acquired changes cannot be made, although these changes are confined strictly to the cochlear duct and the saccu'e. The previously described evidence of a low grade middle ear infection in childhood makes a spread through the round window into the inner ear a definite possibility. The probability of such a spread should not be overestimated, however, in view of the great frequency of such middle ear changes and the great rarity of simultaneous pathologic changes in the inner ear. The presence of deafness in both parents and in many members of both families seems to outweigh in importance the possibility of a bilateral tympanogenic inner ear infection and to favor the assumption of an inherited deafness.

The inner ear changes in case 2 could also be regarded as "post-hydropic," with complete secondary collapse of Reissner's membrane and the saccule. The stria vascularis, again, contained concrements in some of the vessels. In addition, however, hyaline globules were found within the striate epithelium, in the epithelial strings traversing the lumen of the cochlear duct and among the degenerating remnants of the organ of Corti. They evidently represented degeneration products of epithelial cells.

The thinness of the osseous framework of the cochlea has already been observed in other cases. Together with changes in the epithelium of the cochlear duct, it was regarded by Siebenmann as a characteristic feature of the so-called typus Scheibe of deaf-mutism. It is difficult to conceive of its representing an acquired change connected with the supposed anomalies of the endolymph. It was more likely an additional

developmental anomaly, a resorption of the perilymphatic tissue during the stage of cavity formation slightly beyond the normal limits. The formation of the labyrinthine capsule is known to take place under the inductive influence of the epithelial anlage. If a part of the epithelial anlage, the cochlear duct, shows anomalies, the inductive influences emanating from it and acting on the surrounding mesenchyme could also easily be anomalous and induce an excessive resorption of the mesenchyme. The cases of Scheibe's type represent an intermediate stage between those with the changes confined to the epithelial structures of the pars inferior and those with marked anomalies of the entire cochlea.

The crista in the medial wall of the superior sinus of the left utricle was, apparently, an "atypical epithelial formation" (Alexander, 1902), which is not infrequently present in this area. These formations are regularly observed in embryos but later disappear more or less completely. According to Fischer, they do not represent vestiges of the macula neglecta of the lower vertebrates, as assumed by many authors, but their morphologic significance is unknown. In very rare instances vestiges of the macula neglecta are found in man, but they occupy a different position.

In view of the absence of clinical data, it must remain undecided whether this case represents an inherited or acquired deafness. The absence of inflammatory residua; the thinness of the osseous framework of the cochlea; the bilateral and equal involvement of both cochlear ducts, and the freedom from changes of the pars superior of the labyrinth would, however, in this case also, favor the assumption of an inherited deafness.

Considering the very great diagnostic difficulties, it does not seem surprising that Fähndrich in 1935 could collect from the literature only 4 cases in which an acquired form of deaf-mutism could be excluded with certainty and in which, according to the history, a recessive hereditary trait was probable (Scheibe, 1895; Oppikofer, 1903; Siebenmann, 1904; Alexander, 1905). In 4 other cases the deaf-mutism was combined with retinitis pigmentosa (Siebenmann and Bing; Alexander, 1919; Nager, 1927, 2 cases). Four additional cases of probable recessive deaf-mutism were reported by Albrecht (1937, 1939), and several others, such as those reported by Görke, Quix and Brower, Lange, Mayer (case Treumert), Hofmann (1923) and Alexander (1925), could belong to the same group.

Recessive or sporadic deaf-mutism is not infrequently combined with other hereditary defects, not only, as just mentioned, with retinitis pigmentosa but also with imbecility, retinitis pigmentosa with pituitary and skeletal anomalies (Laurence-Moon-Biedl syndrome) and others.

The most important, and perhaps the most frequent combination, is that with the recessive form of retinitis pigmentosa (Bell, Yamada, Steinberg, McGovern, Biró and others).

The histologic observations with regard to the cochlea in the 4 cases mentioned are essentially identical with those in the cases without retinitis pigmentosa. It should be mentioned, however, that in addition there is present a pronounced atrophy of the terminal branches of the labyrinthine vessels, particularly in the cochlea. In the case of Siebenmann and Bing, a lateral dislocation of the organ of Corti in the basal turn also was observed. The sensory cells of the maculas and cristae were normal in Nager's cases; slightly atrophic in Siebenmann's case, and markedly atrophic in that of Alexander. The varying degree of degenerative atrophy of the maculas and cristae explains the fact that the vestibular apparatus in these cases presented either normal, decreased or even no responses.

The examination of the brain in the case of Siebenmann and Bing and in case 1 of Nager showed a pronounced atrophy of the primary acoustic centers (ganglion ventrale and tuberculum acusticum); the atrophy decreased toward the cerebral cortex (Heschl's convolution).

In other instances retinitis pigmentosa was combined with varying but lesser degrees of perception deafness and a gradual loss for the successive higher frequencies. Reports of histologic examination in such cases are, however, lacking.

Many authors assume that recessive deaf-mutism, retinitis pigmentosa and other hereditary defects are due to a common hereditary factor (Siebenmann and Bing, Nager, Hanhart and others). Albrecht and his school denied such a connection and pointed out that retinitis pigmentosa might also be found together with the dominant type of deaf-mutism (Vogelsang, Reich and Barth, Harms-cited by Weber-and Schütz). They regarded the combination as accidental, perhaps favored by gene linkage. In a side branch of a large family with hereditary sclerosis, spinal form (Friedreich's ataxia), in many members and recessive retinitis pigmentosa or retinitis punctata albescens in others, dominant deaf-mutism was found through four generations by Franceschetti and Klein. These two authors did not agree, however, with the strictly "dualistic" conception of Albrecht and his school and advocated a common hereditary factor in spite of the differences in the hereditary traits.

### REVIEW OF THE REPORTS OF CASES WITH EVIDENT DEVELOPMENTAL ANOMALIES

Cases comprising the second main group were characterized by the presence of decided developmental anomalies, not only of the cochlear duct and the saccule but also of the osseous framework of the cochlea.

Siebenmann named the cases "typus Mondini" because a structure was present that was more or less similar to that described macroscopically by Mondini (1791) and later microscopically by Alexander (1904). In all of them, both sides were affected. The bony capsule of the cochlea was flattened from the base to the apex, and the normal arrangement of the scalae was noted only in the basal coil where the septum between the lower basal and the middle turn, originating from the cartilaginous capsule, is present. In the upper parts, the interscalar septums, formed through intramembranous ossification within the connective tissue, were absent and a scala communis (Alexander), or cloaca (Nager), was formed. The modiolus and the spiral laminas also showed defects of varying degree. The cochlear duct often presented a reduction in the number of turns to two or even to one and one-half. Its lumen was, in some instances, markedly dilated, either along the entire course or in its upper parts only. In other cases, the duct had collapsed. The saccule and the endolymphatic duct and sac were often dilated. In other cases, however, there was collapse of the saccule. The utricle was also dilated sometimes, for instance in the case described by Albrecht (1944). The semicircular canals were, as a rule, normal, although malformations in these parts have been described in which, in addition, a congenital atresia was present (Altmann). The aqueductus cochleae may also have been partially or completely absent (Nager, Altmann). The organ of Corti was usually absent in a part of the duct, particularly in the more malformed upper region, and was reduced to a flat mound of more or less undifferentiated cells in other parts. In some instances it showed typical "posthydropic" changes (Altmann). The stria vascularis and the ligamentum spirale were often somewhat atrophic. Frequently, the spiral ganglion presented a reduction in the number of ganglionic cells and nerve fibers belonging to the more affected upper parts; in rare instances it was completely absent. In other cases the ganglion could be noted situated in the central parts of the modiolus and in the fundus of the internal auditory meatus instead of in the usual spiral arrangement. The cochlear division of the eighth nerve was often abnormally thin. A decided reduction in the number of ganglionic cells of the primary cochlear nuclei had been reported by Gauger in one case and by Albrecht (1944) in another, together with developmental anomalies of the cells.

Cases belonging to this group have been described by Mondini, Mackeprang-Ibsen, Alexander, Alt, Iwanow, Lange, Mayer, Neumann, Brunner, Nager, Fraser, Albrecht (1933), Rainer, and Albrecht (1944).

Similar changes had been noted by Alexander and Tandler in the cochlea of 2 congenitally deaf dogs.

Cases in which the malformation of the inner ear was combined with a so-called atresia of the external auditory meatus were reported by Krampitz, Alexander and Benesi, Precechtel, Hofmann, Altmann and Cantele.

The reduction in the number of the cochlear turns, the anomalies in the arrangement and the defects or sometimes even the complete absence of the spiral ganglion were evidently due to impeded development of the cochlear duct and the spiral ganglion. The anomalies of the modiolus, of the interscalar septums and the spiral laminas were due to disturbances in the extent of resorption of the perilymphatic tissue and the ossification within this tissue, and in the arrangement of the osseous structures.

The anomalies of the mesenchymal parts in these cases were probably dependent in their extent on the degree and nature of the malformation of the ectodermal parts. It should, however, be kept in mind that anomalies in the mesenchymal parts were often found in the absence of anomalies in the epithelial or nervous elements of the cochlea (Guild, Polvogt and Crowe).

In view of the anomalous development of the cochlear duct and of the spiral ganglion, there is a definite possibility that some of the changes in the epithelial structures, particularly the organ of Corti and the stria vascularis, might also have been of developmental origin. The absence, for example, of the organ of Corti in the more malformed upper parts and its reduction to a flat mound of cells in the lower parts has already been regarded by various authors as hypoplasia and early arrest of development. It cannot be denied that such developmental anomalies could have been present in some of the cases, particularly in those in which the changes were more pronounced in the underdeveloped upper parts. In many instances, however, the changes were essentially the same as in the previously discussed group of cases. Again, the changes could very well have been the result of anomalies in the cochlear endolymph which might have produced the dilatation frequently noted in the cochlear duct and the saccule; the cases with and without dilatation would represent various degrees of the same type of malformation. The finding of typical "posthydropic" changes together with decided dilatation of both cochlear ducts in a full term fetus with bilateral Mondini malformation, reported by me in a previous paper, can be taken as an indication that the endolymphatic disturbance was already present during fetal life. Albrecht (1944) expressed the belief that the changes could be caused by a fetal meningogenic serous labyrinthitis; Secrétan assumed a primary malformation of the endolymphatic sac with a disturbance in the resorption of the endolymph, causing a dilatation of the sac and of the parts above it. The endolymphatic duct and sac are, however, enclosed in bone or dura, and their dilatation

could hardly have been caused by an increase in the endolymphatic pressure. Such an increase would have had to take place before formation of the osseous labyrinthine capsule, but this would have been long before the time of differentiation of the endolymph-producing structures. It is, therefore, more likely that the dilatation represents a coordinated malformation, an excessive growth, of the endolymphatic appendage. The presence of a disturbance of the resorption in the sac is still not proved; even if present, it would not, however, satisfactorily and invariably explain the dilatation of the higher parts.

Although Ohma observed dilatation of the saccule and the cochlear duct together with occlusion of the endolymphatic duct, the presence of these conditions is by no means the general rule. Incision, cauterization, application of pressure (McNally) or complete destruction of the endolymphatic sac (Lindsay) in animal experiments has failed to produce changes in the saccule or cochlear duct. This failure is explained by the results of morphologic studies of Fieandt and Saxén and of experiments of Altmann and Waltner, which seemed to indicate that the resorption at least of the cochlear endolymph occurs to a large extent in the cochlea itself and not in the endolymphatic sac. The fact that in most of the observations the pars superior was free from dilatation could be explained in the same way as in the previous cases by an anomaly of the endolymph production confined to the pars inferior or by the presence of the valve described by Secrétan.

In some of the cases in question, even with dilatation of the cochlear duct, considerable hearing ability was still maintained. This indicates that the anomalies of the framework and of the length and the lumen of the cochlear duct alone are not a sufficient explanation for the total amount of hearing loss. Such anomalies were described by Polvogt and Crowe in patients with normal hearing who were observed before death. The determining factor seems to be the condition of the stria vascularis, the organ of Corti, the spiral ganglion and the other parts of the cochlearis system. The extent of the changes in these parts determines the degree of hearing loss.

Clinically, malformations of Mondini's type may therefore show complete deafness or only a partial loss of hearing. The partial loss of hearing presents the characteristics of a perception deafness, but in accordance with the fact that the upper parts of the cochlea are the main seat of the malformation, hearing for the low notes is, as a rule, more affected than that for the high.

Malformations of Mondini's type are regarded as the anatomic basis of certain cases of the so-called hereditary degenerative type of impaired hearing and deafness. The deafness shows, according to Albrecht and his school, a dominant hereditary trait, an assumption which has been widely accepted by geneticists (Verschuer).

CASES WITH PROBABLE PRIMARY CHANGES IN THE SPIRAL GANGLION

Hereditary degenerative deafness comprises, however, not only the rarely observed complete or at least marked loss of hearing but also progressive deafness, abnormal fatigability and susceptibility to wear and tear and to acoustic traumas, premature presbycusis and similar conditions, occurring in a much larger group. The hearing loss in this group ranged from a hardly noticeable loss to complete deafness. In cases reported by Helweg-Larsen and Ludvigsen, the diminished hearing was combined with congenital familiar anhidrosis. Both defects were transmitted by simple dominant inheritance. The underlying anatomic changes are not well known. It seems highly probable, however, that they consisted, in analogy with the observations of Crowe, Guild and Polvogt and of Saxén on presbycusis, mainly in degenerative changes in the cells of the spiral ganglion and the nerve fibers but without involvement of the organ of Corti and the stria vascularis. The atrophy of the ganglion which affected the basal parts more than the higher parts could be explained as a "localized organ weakness of the cochlear system," as expressed by Streeter. The ordinary differences in tissue quality observed in the normal person intergrade with cases in which the differences are extremer and of more serious consequence. The embryologic history of an organ composed of tissues of inferior quality seems to depend on the extent of this inferiority and on the nature and time of incidence of various unfavorable environmental factors. Thus, a given combination of these factors may bring about an early arrest of development, resulting in a congenital defect. At the other end of the scale appear various degenerative processes in the inner ear, in the retina and in various parts of the central nervous system in later life ("Aufbrauchkrankheiten"—Edinger, Seligmann). The relation between the nerve endings in the organ of Corti and the hair cells is not yet definitely clarified. It seems, however, that the hair cells can remain intact for a long time, even after extensive degeneration of the cells of the spiral ganglion and of the nerve fibers. The peculiar distribution of the nerve fibers in the organ of Corti whereby each hair cell, particularly each outer hair cell, is innervated by more than one nerve fiber (Lorente de Nó) might play a role in this peculiar behavior. In other instances, however, degeneration in the spiral ganglion and the nerve fibers was followed by degeneration of the organ of Corti. This can be concluded from the findings of Kolmer and, particularly, of Cohrs in horses. These authors frequently observed degenerative atrophy of the cells of the spiral ganglion and of their central and peripheral processes in the basal and middle turn together with degenerative atrophy of the cells of Corti's organ in the same area. In Corti's organ, the changes started with degeneration of the hair cells and led, finally, to replacement of the entire specific neuroepithelium by a single

layer of flat indifferent cells. The stria vascularis was moderately atrophic in some instances but not in all; all the other parts of the cochlear duct remained unchanged. Residua of inflammatory changes were completely absent. There was a close parallel between the intensity of the changes in the spiral ganglion and the intensity of the changes in Corti's organ. The saccule, utricle and semicircular canals appeared normal. The changes seemed independent of sex, race and color of the hair of the horses. They were, according to Cohrs, probably produced by various factors on the basis of an inherited disposition.

## SUMMARY AND CONCLUSIONS

In the great majority of the cases of inherited deafness changes are seen in the organ of Corti, the stria vascularis and, to a much lesser extent, in the cochlear nerve fibers and the spiral ganglion cells of the basal portion of the cochlea. In addition, a degenerative atrophy of the macula sacculi is often observed in man, dogs and cats but rarely in rodents. The primary acoustic nuclei and the central acoustic pathways are normal in some and atrophic in other instances. The utricle and the semicircular canals are always free from changes.

The cochlear duct is either completely or partially collapsed; ectatic or of normal width, or it shows a combination of these conditions. Histologically are noted various degrees of degenerative atrophy of the organ of Corti and of the stria vascularis, or a picture of "posthydropic neuroepithelial degeneration" or a combination of both conditions. The changes are either uniform or more pronounced in the basal than in the apical parts of the cochlea. The changes have been explained as the result of an inherited anomaly of the cochlear duct manifesting itself primarily in an anomaly of the secretion or composition of the cochlear endolymph. In most of the cases the changes in the cochlear nerve fibers and in the spiral ganglion are probably secondary to those in the organ of Corti. The changes in the primary acoustic nuclei and the central cochlear pathways are not directly connected with those in the cochlea but are a coordinated anomaly, either a primary hypoplasia or an inherited organic inferiority with secondary degenerative atrophy.

Sometimes the aforementioned cochlear changes are combined with an abnormal thinness of the osseous framework. This condition was explained as the sequel of abnormal inductive influences emanating from the anlage of the cochlear duct and acting on the surrounding mesenchyme.

Two cases of probable inherited deafness are reported, one of them without and one with changes in the osseous framework of the cochlea. The difficulties of a histologic differentiation of cases with inherited and with acquired deafness are pointed out and discussed.

In all cases of inherited deafness a recessive hereditary trait is present. It is not infrequently combined with other hereditary defects, for instance, retinitis pigmentosa.

The cases with decided macroscopically visible anomalies of the cochlea when fully developed present a flattening of the cochlea from the base to the apex with extensive defects in the parts of the osseous framework formed by intramembranous ossification. The number of cochlear turns is often reduced and the cochlear duct dilated, either in its entirety or in the upper parts only. The saccule is often dilated, the endolymphatic duct and sac frequently abnormally wide. The organ of Corti may be completely absent, sometimes in the upper parts only. If present, it may show, together with the stria vascularis, the same changes as in the previously discussed cases. The spiral ganglion is either completely absent, abnormally located or more or less atrophic, particularly in the upper parts. Hypoplasia of the primary acoustic nuclei in the brain stem has been found in the 2 cases reported on.

The changes in this form of inherited deafness are more distinct and involve not only the cochlea and saccule but, as a rule, also the endolymphatic duct and sac. Within the cochlea they show a greater intensity in the upper than in the lower parts. The observations can best be explained by an anomaly of the epithelial anlage, severer than in the preceding cases and producing malformations of the cochlear duct and the endolymphatic appendage. The changes within the cochlear duct can, in many instances, again be explained by an inherited anomaly of the secretion and composition of the cochlear endolymph. The epithelial changes are, however, often more pronounced in the underdeveloped upper parts of the duct. This condition could indicate that, at least in certain instances, not only degenerative atrophic and "posthydropic" changes but also developmental anomalies of the epithelial parts might have been present. The anomalies in the osseous framework are again secondary to those in the epithelial anlage. The changes in the spiral ganglion and the cochlear nerve fibers might be secondary to those in the organ of Corti in some instances but are unquestionably of developmental origin in others. Together with the changes in the primary acoustic nuclei they then represent coordinated anomalies.

These malformations of "Mondini's type" are regarded as the anatomic basis of certain cases of inherited deafness with a dominant hereditary trait in man. Similar changes have been described in dogs.

The anatomic changes underlying the large group of cases with progressive deafness, abnormal fatigability and susceptibility to wear and tear and to acoustic traumas with premature presbycusis and similar conditions are not definitely known. They are, however, most probably different from those in the preceding cases and seem to consist primarily in degenerative changes in the cells of the spiral ganglion,

particularly in the lower parts, on the basis of an inherited "localized organ weakness of the cochlear system," as expressed by Streeter. The hereditary trait is dominant in some instances but undetermined in the majority of them.

In conclusion, therefore the anatomic findings in inherited deafness can be divided into two types.

1. Primary changes in the cochlear duct and saccule: (*a*) changes confined to the epithelial parts of the cochlear duct with or without involvement of the saccule (so-called sporadic deaf-mutism in man, deafness in dogs and cats with pigment anomalies, deafness associated with locomotor anomalies in rodents), (*b*) additional slight anomalies of the osseous framework of the cochlea (in man) and (*c*) marked anomalies in the membranous and osseous parts of the cochlea, the saccule, the endolymphatic duct and sac (so-called typus Mondini in man and dogs).

(In all the subgroups, particularly the last-named one, coordinated anomalies of the central cochlear system might be present.)

2. Primary degenerative atrophy of the spiral ganglion on the basis of an inherited "localized organ weakness of the cochlear system" (man, horses).

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Prof. Dr. Emil Schlander, Head of the Ear, Nose and Throat Department, University of Vienna Medical Faculty, permitted the study and publication of the series.

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# TRANSANTRAL APPROACH FOR EXCISION OF SPHENOPALATINE GANGLION FOR INTRACTABLE FACIAL PAIN

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FRAZIER, three decades ago, expressed the hope that a simple approach might be elaborated for the excision of the sphenopalatine ganglion. He was highly impressed by the beneficial effect derived from an anesthetic drug deposited on or near the ganglion. To his mind, however, the amelioration obtained this way was, unfortunately, temporary; permanent relief from the excruciating pains could be expected only after the sphenopalatine ganglion had been excised.

Since the preceding statement goes directly to the theme of this paper, it should not be passed off without some serious reflection. Frazier observed that certain persons during their period of suffering obtained swift and gratifying alleviation when an anesthetic drug was brought into contact with the nasal ganglion. The drug might be carried on a pledget of cotton or on an applicator or might be injected. There were undoubtedly times when the drug was not brought into immediate contact with the body, yet amelioration of the excessively severe pain took place. From descriptions given, it is safe to assume that the drug was deposited in the neighborhood of the ganglion, and its action was probably effected by diffusion. Relief was brought not only to those patients suffering mild pain but to those experiencing agonizing tortures. This therapy recommended itself not only because of its simplicity of application but also because of its offering more in the way of mitigation than any other measure with which Frazier was familiar. Indeed, even gasserectomy, the procedure that generally offers the widest range of relief to persons suffering torture in the face and head, failed in some instances after it was performed. In some cases, the interruption of the continuity of the fifth cranial nerve and the stripping of the sympathetic nerves from the carotid arteries failed to bring relief. And yet, when seemingly unsuccessful with such major surgical endeavors, his efforts

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were more successful when he applied cocaine to the sphenopalatine ganglion. The remissions, unhappily, were of only short duration. The cocaine could be and was applied again, but the improvement therefrom was again of short duration. These experiences induced Frazier to wish for a simple operation to excise the ganglion. Therein, he thought, lay the hope of bringing more permanent relief to tortured victims. I shall briefly cite an example from Frazier's cases:

The patient, aged 30, was operated on for excision of the gasserian ganglion. Three years afterwards he returned complaining of as much pain as before, referred almost altogether to the region of the mandible. The superfacial cervical plexus was excised but to no avail. There were a steady pain in the right lower alveolus and paroxysms of electric-like pain which shot into the alveolus and angle of the mouth. Ciliospinal paralysis and angioneurotic edema of lips and eyelids occurred. The paroxysms might be started by apprehension of interference, by scraping, rubbing, pinching or sticking of the face or adjacent cervical areas or by pressure over the right cervical portion of the sympathetic trunk. There had been recently ciliospinal paralysis with angioneurotic edema of the lips and eyelids. Because of the latter phenomenon, because of the tenderness over the cervical portion of the sympathetic trunk and because of the apparent existence of afferent fibers (paroxysm of pain being excited by rubbing, pinching or sticking of the face), denudation of the arterial plexus of the common and external carotid arteries was proposed. This operation was practiced but with no appreciable effect on the pain.

### OPERATION

The technic of the transantral approach for the excision of the sphenopalatine ganglion is relatively simple. Those persons trained in rhinologic surgery will find the operation particularly easy to perform.

After the customary preoperative administration of a sedative, and the preparation of the operative field, a 1 or 2 per cent solution of procaine hydrochloride mixed with a minimal quantity of epinephrine hydrochloride is infiltrated beneath the soft tissues of the upper lip and cheek of the side to be explored. The infraorbital nerve at its exit from its foramen is also infiltrated. A needle is introduced through the large palatine foramen and extended upward about 1½ (4 cm.) inches, and approximately 2 cc. of the solution is deposited to anesthetize the group of nerves with the ganglion in that region. If the surgeon should elect to anesthetize the ganglion with its connections by applying the drug on an applicator introduced by way of the nose, no objection can be raised to the maneuver. My own preference is for the method of infiltration. The area is now insensitive. Next, a lengthy incision is made close to but not at the line where the mucosa of the alveolar process passes over the cheek. The soft tissue is elevated; the bony parts of the maxilla are exposed, and the infraorbital nerve is completely outlined as it emerges from its foramen. A small piece of the antral wall is taken away; this serves to expose the maxillary antrum. The anterior wall should be pierced through the thin portion of the plate on a rather high plane. The rest of the bone is then removed in an upward direction until the nerve is well outlined in its canal. A needle is thrust into the canal and advanced posteriorly, the lightest touch being used to avoid piercing any obstacle that might be lodged in the canal, and a small quantity of procaine hydrochloride solution is deposited. The entire field of opera-

tion should now be completely insensitive to subsequent acts. An incision into the mucous membrane covering the floor of the bony canal is made with a sharp instrument and the membrane elevated; then, with a fine chisel, the bone forming the floor of the infraorbital canal is removed as far as the posterior wall. This procedure exposes the nerve in its extent from the soft tissues of the cheek to the posterior wall of the antrum. A small portion of the bony posterior wall around the nerve is then carefully removed by means of a sharply pointed instrument. When this is carefully done, the field is entirely dry. Only soft tissue now surrounds the nerve; it is carefully separated from around the nerve, and the sphenopalatine ganglion is exposed. Liberation of the nerve is continued beyond the ganglionic mass, near to the foramen rotundum. The nerve is taken out of the infraorbital canal with a small instrument similar in construction to a dental explorer or with a nerve hook. The nerve now lies free in its entire extent from the foramen rotundum posteriorly to the cheek anteriorly. The ganglion is freed and detached from all its connections except the infraorbital nerve. The infraorbital nerve is divided close to the foramen rotundum beyond the ganglion and taken out in its entirety with the sphenopalatine ganglion and detached from the soft tissue at the cheek. Thus a large portion of the maxillary nerve, the sphenopalatine ganglion and the tiny tendrils connecting the two are removed in one sweep. The small infraorbital vessels travel with the nerve. Surgical care must be exercised in division of the nerve in the pterygopalatine fossa or bleeding of annoying nature may take place. If slight traction is played on the nerve and a very fine artery forceps placed on its body and sparked with an electric current, bleeding will not occur when division is accomplished. Finally, a strip of gauze can be placed in the operative field with one end brought out of the mouth. The soft tissues at the alveolar process and the cheek can now be sutured. In a day or two the gauze is removed. Sutures are removed after several days.

## REPORT OF CASES

The foregoing technic was used in the following cases.

Case 1.—A. W., a 54 year old woman, was admitted to the hospital on Oct. 21, 1948. She gave her history as follows: She had had von Recklinghausen disease for several years. Some small tumors were removed from her arms, and a histologic examination established the nature of the disease; her present physician confirmed the diagnosis. She had been troubled with distress of the face and head for several years before the present admission; the cause was thought to be inflammation of the accessory nasal sinuses, and an operation on the left nasal antrum was performed four or five years before this admission. She had some improvement at first; then the distress increased with greater severity, especially during the winter. Disturbances such as drafts of wind striking the face, unusual noises—loud voices, scraping, drilling or hammering—an unexpected movement of the head or body or a drink of cold or hot water set off a chain of lightning-like piercing strokes which began at the angle of the nose, continued through the face and the front and back of the head and finally centered in the back of the eye, where there was a terrific boring pressure and a feeling as if her flesh were being torn. There was some compression in the cheek extending to the ear. On examination, the patient's distress was readily discernible. She had eaten very little. Therefore, a sphenopalatine ganglion block was made instantly in order to afford relief. Three cubic centimeters of a 2 per cent solution of procaine hydrochloride was introduced into the sphenopalatine ganglion by means of a needle passed

through the palatine foramen. The patient's pain was relieved, and the examiner was able to look into the nose and mouth and to palpate the apparent sites of the seizures. During this procedure, the examiner's fingers brushed the soft tissue overlying the left nasal antrum, and she gave a loud scream of pain. She was hospitalized and a full study was made. A roentgenogram of the nasal accessory sinuses showed evidences of a cloudy left antrum. Irrigation of the antrum revealed nothing significant. There was irritation of all three divisions of the trigeminal nerve. The second maxillary nerve showed definite evidence of involvement, while the condition of the ophthalmic and mandibular nerves could be classified as borderline. Since a consultation with the neurologic department was necessary, Dr. J. Arthur MacLean, neurosurgeon of the University Hospital of the New York University–Bellevue Medical Center, and I examined the patient. We agreed that we were dealing with a region served mainly by the maxillary nerve, and therefore I should perform the transantral operation for the resection of the sphenopalatine ganglion and portion of the maxillary nerve; if this procedure failed, Dr. MacLean would perform a gasserectomy. The patient's family consented to the operation. The ganglion was resected, and the patient was relieved instantly. Nine months postoperatively, I examined her again. During the interval, she had remained free of the pains, and at the time of writing she reports no recurrence of these pains.

During the operation, granulation filling the antrum was removed. This granulation tissue may have been responsible for the distressing symptoms, and its removal would perhaps have freed the patient from pain. This possibility was considered in detail. Evidence gathered from the patient, her doctor, consultation with the neurologist and my own experience leads me to the conclusion that had the ganglion and the portion of the infraorbital nerve been left the patient would undoubtedly have continued to suffer, without the slightest abatement, in the same degree as she did before the granulations were removed.

Case 2.—M. M., a white man, 36 years old, reported that in an accident several years previously his head had been caught between the wall of an elevator and the elevator shaft. Although he had never suffered from headaches before this accident, he now complained of grueling headaches. He had had medical attention and had undergone several operations. He was referred to me by his oral surgeon.

The patient complained of disabling pains having their trigger area in the upper alveolar process near the lateral incisors of both sides and fanning out in all directions. He wished to study law and was therefore working in a law office, but the pains made working and studying impossible. Examination disclosed that the mouth was toothless. Although he possessed a complete denture, he seldom wore it because of caustic aches. He was referred by the oral surgeon for excision of the nasal ganglion and a portion of the infraorbital nerve to afford relief.

An operation for removal of these organs on the left side was carried out, and the patient felt comfortable thereafter. Encouraged by the favorable result, he made arrangements for the excision of the ganglion on the right side. When the maxillary bone was exposed after incision and elevation of the cheek, it was discovered that a major portion of the lateral and posterior walls which form the boundaries of the antrum of Highmore were absent and that the soft tissue not only had replaced the bony structure but had almost completely obliterated the antrum. When the posterior boundaries of the infraorbital nerve in the roof of the antrum were removed to expose the pterygopalatine fossa, the landmarks were not

readily identifiable. The nerve, however, was divided as it emerged in the pterygopalatine fossa into the infraorbital canal. The portion filling the canal was removed. The execution of this procedure did not give entirely satisfactory results. A short time after completion of the operation, the patient returned to the clinic and complained of a pinching sensation in his mouth. Chewing and talking aggravated the discomfort and he was not able to wear his denture. However, at this time the disagreeable sensation was more in the nature of an aggravated discomfort than a disabling pain, and consequently, despite his pleas, further surgical treatment was denied him at the time. Later, an operation was undertaken through the mouth. The large palatine foramen was exposed and the bone posteriorly removed. The palatine nerves were exposed superiorly and divided, and about 1 cm. was snipped off. The patient's progress was followed for six months. His complaints were singularly few. Several recent attempts to locate him to check on his present status failed. The left side of the head and face remained consistently free from the original distress and it would be interesting to know the present condition of the right side of his face and head.

Case 3.—R. T., a woman aged 51, was admitted to the hospital on Sept. 1, 1942, with an uncomfortable feeling in the face, particularly in the right side, which had been present for many years but which did not cause much discomfort. She recalled when she had had the first pang in her nose, a number of years ago. The pain was like an electric shock, but it passed and it was a long time before another startling sting hit. Thereafter they came more frequently. During the past two years, she could not eat or drink properly; consequently she had lost considerable weight and could not sleep or rest. The sharp needle-like pains might strike once and then pass away, or they might strike a number of times consecutively. At first they appeared during the winter and in the summers they were comparatively rare. Lately, the patient was just as uncomfortable during the summers. The pain was like the grinding of points of needles rapidly and forcibly into the flesh. The sharp thrust extended from the tip of the nose to the eye and deep into the head. The pains would then be followed by a cold sweat. The patient had had considerable medical attention.

Treatment consisted in application of cocaine or tetracaine hydrochloride solution to the nasal turbinate bodies and to the sphenopalatine ganglion posteriorly and laterally to the posterior tip of the middle turbinate. A solution of 2 per cent procaine hydrochloride was injected with a syringe and a Ruskin needle through the palatine foramen around the area of the nasal ganglion. The beneficial effects were unfortunately only temporary.

Because of a particularly severe attack of pain, the patient was hospitalized and a complete study made. Results of laboratory and medical studies were essentially noncontributory. The ocular fundi and muscular reflexes were normal. Dr. Carl Peterson, neurosurgeon at the New York Post-Graduate Hospital, was called into consultation. After examining the patient, he was unable to determine whether she had neuralgia of the gasserian ganglion or neuralgia associated with the sphenopalatine ganglion. The second and third divisions of the trigeminal nerve and the area supplied by the maxillary nerve were seemingly involved. Fibers carried by the supraorbital branch were excessively sensitive. Of the three divisions, the least involved was the first; the third appeared to be implicated, and the second was the most seriously affected. We decided to recommend a transantral resection of the sphenopalatine ganglion. If the operation failed, the neurosurgeon would perform a gasserectomy. After consent from the patient was

obtained, the transantral procedure was undertaken. The sphenopalatine ganglion with the portion of the second branch of the trigeminal nerve extending slightly posterior to the ganglion to the infraorbital foramen was excised. The patient has reported no recurrence of pain since the operation.

Case 4.—M. R., a 76 year old woman, mother of eight living children and of three who are dead, reported suffering from severe facial pains for several years. Previous treatment was unsatisfactory. Examination disclosed that the patient was fairly well nourished, short of stature, slightly stooped and had enlarged arthritic joints in her hands and feet. The articular and muscular pains were not severe, but when the facial pains struck all her activities ceased. Dentures had to be removed, and she neglected oral and physical hygiene. Since she was unable to chew, a nutritional deficiency became manifest, and constipation, abdominal distention and gastrointestinal disturbances followed. Trigger points of pain could be elicited around the area of the upper premolars on both sides. Cold water, cool drafts of air, a bite of food or a slight stroking of the cheek brought piercing lightning-like pains to the alveolus of the maxilla that spread quickly to the eye, forehead, cheek, ear and mastoid region. The patient complained of a drawing, gnawing sensation in the arms, tingling in the hands and muscular cramps in the back which often extended down the legs. The mandibular and maxillary regions were also involved.

Dr. Peterson and I agreed on the operation to be tried first. Even though pains could be located in the second division of the trigeminal nerve, involvement of the first and particularly the third division of the nerve could not be ruled out entirely. It was decided that, since considerable relief was attained by an injection of procaine hydrochloride into the area of the sphenopalatine ganglion, the organs located there were probably responsible for the patient's agony. If the results were unfavorable, gasserectomy would be done. The transantral resection of the sphenopalatine ganglion was performed, and a large portion of the maxillary nerve was removed on the left side under local anesthesia. The patient remained entirely free from pain as a result of this procedure. Three months later we attempted the same operation on the right side of the face, but a sudden disturbance of the circulatory system set in; therefore we thought it safer to interrupt the procedure before its completion. The infraorbital nerve was divided in the posterior angle of the antral roof and was removed. The sphenopalatine ganglion was not excised. During the five years of the patient's life following the excision of the infraorbital nerve, she complained of pain on the right side of the face and had to have medical attention. Even though the suffering was acute and sharp, it was not so grave nor so protracted as formerly. The pains were readily controlled with an injection of procaine hydrochloride solution into the ganglion. Had the patient been in better health, another attempt to excise the ganglion would have been justified.

## SYMPTOMS

As one comes to learn the clinical role played by the sphenopalatine complex, it becomes evident how difficult it is to differentiate the symptoms set in motion by some disturbance within its intricate body from those symptoms produced by changes in the gasserian ganglion. Writers who in the past circumscribed the major neuralgias with definite boundaries would find it necessary today to revise these limitations. Frazier, who recognized a definite clinical entity in what he termed as

"major trigeminal neuralgia" the symptoms of which were so characteristic that a diagnosis admitting of no discussion could be made, would find that this belief was untenable when tested by present day experiences. Statements heard in lectures or read in literature that pains which come as a "bolt from the sky and vanish like a shooting star" belong to the gasserian ganglion complex and can be controlled only by dividing the fibers of that organ are not always valid. Nor is the intimation entirely correct that only aches of longer duration and greater pressure are components of the sphenopalatine complex. Attempts to determine which of the two ganglions was responsible for the excruciating pains along a sharp demarcation would lead to repeated error in diagnosis.

For a long time I believed that there was an essential difference in the quality of the pains emanating from one ganglion as compared with those coming from the other. However, after long study, I found that what appeared to be a good formula at one time was inadequate at another. This experience recurred so often that, for the present, I contend that narrow classification of the symptoms will fail if it is tried. The neurosurgeons from the department of neurology, called into consultation in 3 of my cases herein cited, were unable to decide with any degree of assurance which one of the two ganglions was responsible for the suffering. In 2 of the 3 cases, the neurologist could not decide which ganglion was more accountable for the extreme sensitiveness present during the examination. There are many instances in which the resection of the gasserian ganglion was unsuccessful. Gardner recently performed an unsuccessful unilateral prefrontal lobotomy to relieve the severity of pain in his patient. These facts are presented to illustrate how discouraging it is to classify pain symptoms with a view to determining their origin. In my opinion, when difficulty is encountered in deciding whether the gasserian or sphenopalatine ganglion may be the source of the pain which is to be relieved, the latter is my choice for any contemplated surgical intervention. If the results are disappointing, the ganglion may be excised.

I am reporting the results obtained from a series of patients who had undergone surgical intervention which did not consist in the excision of the nasal ganglion alone; part of the maxillary nerve was removed at the same time. The importance of this ganglion in the causation of many forms of atypical disturbances will not be known until the ganglion has been excised alone in a series of properly selected cases. In referring to the ganglion as though it were the only tissue which received surgical attention, I did this to bring the treatment into line with the various treatments often accorded the gasserian ganglion, since portions of the nerve are also excised with it; yet the operation is generally called gasserectomy.

Symptoms which patients report and which are worthy of consideration for the excision of the sphenopalatine ganglion are pains referred chiefly to the orbit, temple and cheek, neck and shoulder and occasionally down the arm to the finger tips. Patients complain of pains in the back, extending down the leg to the foot, and tingling and quivering of toes and fingers. Why discomforts in the back or in the extremities should be influenced by the ganglionic structures in the pterygopalatine fossa is unknown.

The principal tortures experienced by patients are the sharp, shooting, lancinating types experienced in one of the facial nerves, usually the maxillary or the mandibular. The pain is like the boring of a hot iron or the tearing of flesh. It begins lightly, gradually working up to a higher pitch until unbearable ferocity is attained. There is pressure behind the eye—a viselike compression becoming tighter around the temple—which may pass to the back of the neck. At times mastoid tenderness or a sharp sticking in the ear is present. Vasomotor changes are observed in the face. Occasionally associated migraine or preauricular, mandibular or maxillary tenderness appears. Herpes zoster of the face has been seen. The attacks vary in duration—a week or two at first and two or three a year, but as time goes on more frequent and of long duration. During an attack, the pains cause the patients to neglect their health and strength since they are unable to chew, swallow or wash their face for fear of an attack. Patients dread a recurrence of pain even after surgical intervention has brought relief.

### WHEN TO OPERATE

The operation seems to be particularly well suited in cases in which the second division of the trigeminal nerve is involved. It is not always easy to be sure that only the second division of this nerve is affected when one is confronted with a neuralgic facial pain. Frequently there is a wide diffusion of pain. Anatomic boundaries are not respected. It will be observed that in the patients examined by the neurologists, the pains were so widespread that the specialists were unable to say that the pains were confined to the maxillary nerve alone. It appeared that some of the fibers of the mandibular nerve were also certainly involved; branches of the ophthalmic nerve, too, were sensitive, and involvement of the sympathetic nerves could not be ruled out. Therefore, care must be exercised before a decision is made to submit a person to surgery. The greatest evidence that the otolaryngologist is duly cautious is that he first uses all conservative means known to bring relief before attempting the more radical. It is only after the various local applications or other treatments of conservative nature have fully proved themselves unsatisfactory and the patient evinces a desire for a more

radical undertaking that this operation should be carried out. The advantage which this method offers over the commoner ones is important; namely, in order to reach the nasal ganglion, the surgeon is not obliged to cut important tissue, in fact, tissue customarily accepted as vital is totally avoided. Grave complications are less likely to happen. The entire process can be carried out with such speed and simplicity as to place it justifiably in the category of minor surgery. If the excision of the sphenopalatine ganglion fails to afford relief, no known damage has been done other than negligible shock attributable to minor surgery, and it in no way interferes with any measure desired to be instituted subsequently. One safe and reasonable way to select patients for this operation is to anesthetize the sphenopalatine ganglion with a drug carried to the organ on an applicator by way of the nose or to infiltrate the area with a 2 per cent solution of procaine hydrochloride, and if the pain vanishes it may be safely assumed that the maxillary nerve or the sphenopalatine ganglion or both are responsible for the distressing clinical manifestations. Clinical experience has indicated that by the excision of the organs permanent freedom from pain may be expected.

Occasionally it is questioned whether equally good results may not be obtained by interrupting the continuity of the infraorbital nerve somewhere in its canal or even after it leaves its anterior foramen. It is my belief that reports of such nature have been made. My own experience has not been fortunate. I have also practiced avulsing and even removing the entire nerve found in the canal extending from the posterior boundary to and including the anterior boundary in the roof of the maxillary antrum; the results have been discouraging. It was, in the main, these failures that led me to investigate the surgery of the ganglion.

The case histories of the patients reported have been given in detail to permit the reader to view the symptoms that were beneficially influenced after the ganglion and portion of the nerve were excised. As to what per cent of the patients may profit from this operation, if a large number should be investigated, is naturally difficult to predict. Its advantage over some of the operations generally undertaken to ease facial neuralgia is the simplicity of the procedure while at the same time exposure and manipulation of the vital tissue are avoided.

## SUMMARY

The comparative lack of specific control measures for facial neuralgia and the wide and frequent occurrence of these diseases have long posed a challenge to preventive medicine.

A new approach to the surgical excision of the sphenopalatine ganglion with its connections is advanced. It is an elaboration of a procedure

reported fifteen years ago. It has the following advantages: It is simple to execute; probable complications following surgical intervention with this approach are at a minimum; vital structures are not encountered; it in no way interferes with or makes more difficult other standard procedures should they be found necessary; patients remain free from pain. Patients have remained free from pain from one to six years and were still without pain at the time that this report was written. This approach was used in operations performed at the New York Post-Graduate Hospital.

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# OTOSCLEROSIS

## Résumé of the Literature with Clinical Observations

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LONG BEFORE otosclerosis was recognized as a separate disease it was considered by otologists as a form of "dry middle ear catarrh." In the middle of the last century, Tröltsch first expressed his opinion that the condition was not a form of catarrh of the middle ear but must be regarded as an entity in the diseases of the ear.

Politzer, pioneer in otology, studied a large number of patients with this condition at the Vienna General Alms House, both during life and at autopsy, and was the first to describe otosclerosis as a primary disease of the labyrinthine capsule. He described otosclerotic foci as proliferating bony growths occurring in certain areas of the capsule only and referred to them as "sites of predilection." He said that as long as the disease lies within the bony labyrinthine capsule, no clinical symptoms could be observed in the patients; but when it extended beyond the endosteal or periosteal layer of the capsule ankylosis of the stapedial footplate occurred, which, in turn, led to the sound-conducting lesion with a normal drum membrane and normally functioning eustachian tube. It is amazing that such a plain and exact statement was made by him as early as the last century.

In 1837 Toynbee described the real pathologic condition as thickening of the footplate of the stapes, which was ankylosed by a bony overgrowth at its anterior two thirds, originating in the vestibular surface of the labyrinth.

In the summer of 1893 Politzer read a paper before the Pan-American Congress at Washington on the pathologic process of otosclerosis, as studied by him at autopsy in a dozen cases. In most of these cases the mucous membrane in the middle ears did not show any change from the normal, and sections of the decalcified bones showed a sharply defined lesion in the region of the fenestra vestibuli. The lesion was

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This paper was made during my six months of service, from March 1 to Sept. 30, 1946, at Dr. Julius Lempert's Institute of Otology, New York.

Thesis submitted to the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science [M.SC. (Med.)] for graduate work in otolaryngology.

differentiated from the normal bone by its deep carmine-staining property; it showed pronounced enlargement of the bone spaces and haversian canals. Politzer's original pictures illustrating the pathology of otosclerosis are shown here.

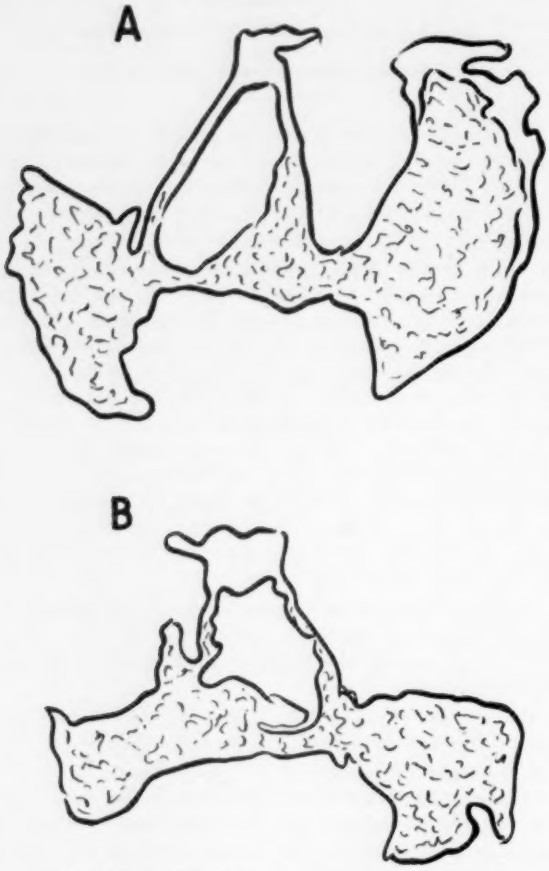


Fig. 1.—Copies of the original pictures illustrating the pathology of otosclerosis presented by Politzer in 1893 (Politzer, A.: The Anatomical and Histological Dissection of the Human Ear in the Normal and Diseased Condition, ed. 4, London, Baillière, Tindall & Cox, 1902.). *A*, otosclerotic process of bone, involving the angle formed by the footplate of the stapes and the anterior crus. *B*, further advanced stage of the disease. Note the sharply defined plate of bone at the anterior part of the fenestra vestibuli and involvement of the entire labyrinthine wall; at the posterior part of the oval window the bony growth enveloped the external surface of the footplate without reaching the opposite border.

The history of otosclerosis may be summarized in four phases of importance.

First, recognition of stapedial ankylosis (Tröltsch).

Second, recognition of the disease as a primary circumscribed process in the labyrinthine capsule dissociated from demonstrable changes in the tympanic cavity (Politzer).

Third, further development in the micropathology and the formulation of diagnostic methods (O'Mayer, Wittmaack, Manasse, Bezold and many others).

Fourth, surgical treatment of otosclerosis as the only successful method (Sourdille, Holmgren and Lempert).

## SYNONYMS

There is perhaps no other disease in medical history which has so much confusion in nomenclature as otosclerosis; this confusion can perhaps be attributed to the great diversity of opinions on the problem of its unknown cause. Following is a list of some of the terms that I gathered from the literature.

1. Chronic progressive deafness
2. Otitis insidiosa
3. Otoporosis
4. Osteospongiosis
5. Otitis media catarrhalis sicca
6. Ostitis vasculosa
7. Ostitis chronica metaplastica
8. Circumscribed ostitis
9. Spongification of the labyrinth
10. Osteofibromatosis
11. Otitis media hyperplastica
12. Hereditary deafness
13. Proliferous inflammation of the middle ear

## PATHOLOGIC ANATOMY

*Histology.*—Normally, the membranous labyrinth is surrounded by two distinct layers of bone, the endosteal and the periosteal.

The endosteal layer of bone is the labyrinth capsule proper. It is made of dense bone directly derived from the cartilaginous capsule of the otic vesicle of the embryo. According to Manasse, this layer encloses the remnants of original cartilaginous capsule called "interglobular bodies." This layer is made of compact lamellar bone and is comparatively nonvascular.

The periosteal layer encloses the endosteal or cartilaginous bone but is distinct from it. It is made of lamellar bone arranged parallel to the surface and is derived from (*a*) the mucoperiosteum of the middle layer and, probably, (*b*) the osteogenic layer of the dura mater.

These two layers are quite distinct in embryos and in infants. According to some authors, even in adults these two layers are separated by a well marked lymph space.

PATHOLOGIC PROCESS

The otosclerotic lesion is a disease of the bony labyrinthine capsule, and it occurs in localized patches within the otic capsule. It is characterized by sharply defined areas, which stain differentially bluish red with hematoxylin and eosin.

In the early stages the otosclerotic areas are more vascular, with a preponderance of marrow-like cavities containing connective tissue cells. Osteoclasts and osteoblasts are seen at times. Routine examination of the temporal bones has revealed the following sites as the usual ones of the lesion in the otic capsule: oval window, round window, cochlea and the internal auditory meatus. But the favorite site, or the "site of predilection," has been the region in front of the oval window. This is the site where the vestigial structure called "fissula ante fenestrum" is present, and, according to Bast, the otosclerotic lesion surrounding this structure is a secondary invasion.

So long as the lesion remains within the bony capsule of the labyrinth there are no clinical signs or symptoms of otosclerosis; but only when the disease encroaches beyond the endosteal or periosteal layer in the region of the oval window, thereby causing partial or complete bony ankylosis of the stapedial footplate, can a history of deafness be obtained during life suggestive of the otosclerotic process.

The enlargement, or the spread, of the otosclerotic foci has been explained by two different views, both of which lack any definite confirmatory proof. One school of thought attributes the spread to the absorption of the normal bone by osteoclasts, to be replaced by the otosclerotic process; the other school explains it as a tumor formation which destroys the normal bone by its growth.

Through the site of predilection is the oval window, with the resulting type of deafness associated with clinical otosclerosis, one must bear in mind, as shown by Nager, that it is possible to have a nerve type of deafness through the involvement of the cochlear region by the otosclerotic lesion.

A summary of the important pathologic changes follows.

1. Otosclerosis is a disease of the bony labyrinth and occurs as circumscribed patches.

2. There is a sharp line of demarcation between the diseased and the normal bone.

3. The staining property of the lesion is different from that of the normal bone.

4. The size, shape and arrangement of cells usual in the normal bone are absent in the diseased bone. In the diseased bone the cells are coarse, irregular in shape and arrangement and increased in number.

5. Very large medullary spaces are present in the otosclerotic foci, and engorged, newly formed blood vessels can be observed in the medullary spaces in the diseased bone.

6. There are no changes in the bone surrounding the otosclerotic foci.

7. No positive means for diagnosis of the disease exist except postmortem histologic examination.

8. Deafness is a manifestation of the disease only when the process involves the stapediovestibular joint.

## ETIOLOGY

The literature concerning the etiology of otosclerosis reveals the amazing fact that there are perhaps few diseases of the human body which have not been considered as the cause of otosclerosis. To put it in a "nutshell," anything from gray hair to an ingrown toe nail may be predisposing factors in otosclerosis. To discuss all the theories is next to an impossibility, and only a general outline of the important ones will be presented here under classified headings. Last of them all, I venture to mention my view on the subject, based mainly on two facts observed during my six months of service at Lempert's Institute of Otology, when I had the unique opportunity of studying more than 300 cases, both before and after the fenestration operation.

1. *Theory of Inflammatory Origin.*—Fraser attributed the cause to a local inflammatory process of the infective type, beginning as otitis media and affecting the mucous membrane and the mucoperiosteum. The argument presented was that since the ossicles of the middle ear and the promontory are covered directly by the mucous membrane, unlike any other bone in the body, even a very mild infection could affect the bone. But it has generally been observed that the middle ear cavity, as well as the mucous membrane, shows no pathologic change in most of the patients with otosclerosis.

According to Manasse, the otosclerotic process originated in the blood vessels of the compact bone, with the formation of granulation tissue and new bone formation. He called it, therefore, otitis chronica metaplastica. The "blue mantle" is the blue-staining cementum surrounding the new blood vessels which radiate from the otosclerotic foci and which are inflammatory.

Gunther expressed the belief that the blue-mantled vessels serve to draw the calcium from the normal bony capsule and to deliver it to the otosclerotic foci. O'Mayer stated the opinion that the blue mantles do not signify any pathologic situation.

Alexander named otosclerosis "ostitis vasculosa," holding the theory that the large, engorged and newly formed blood vessels are the result of inflammation.

2. *Theory of Traumatic Origin.*—The latest theory of O'Mayer is based on "trauma" as the cause of otosclerosis. He described the bony changes as formation of callus resulting from the healing of spontaneous fractures in the otic capsule. According to him, the trauma is not an external one but is due to pressure exerted in certain directions on the labyrinthine capsule. It is hard to imagine how any such trauma could be caused, and even if it were caused, how it could produce fractures in a small bony mass with such a hard, ivory-like consistency. O'Mayer, through a study of a large number of petrous bones, found many small fissures occurring at the same sites in the various specimens. Histologic study of these fissures revealed their resemblance to the healed fractures of the skull filled with fibrous osteoid tissue.

Alexander reported similar fissures in cases of congenital deafness, and J. Fisher also found these fissures in cases of otosclerosis.

3. *Theory of Neoplastic Origin.*—Another theory of O'Mayer is that otosclerotic foci are benign tumors, which he called "hematoma." These tumors, he stated, grow by expansion, and the resulting resorption of the surrounding bone is caused by pressure of the growth. Similar theories have been advanced by Bruhl, Lange and Fisher.

4. *Theory of Systemic Disease of the Skeleton.*—Many otologists have discussed the resemblance of otosclerotic foci to the diseases of the skeleton, such as "osteitis fibrosa," of von Recklinghausen and "osteitis deformans," of Paget. Fisher published a photomicrograph from a case of osteitis deformans (Paget's) affecting the bony labyrinth of the ear, where the disease was found to be widespread, the entire inner ear being involved.

Osteopsathyrosis associated with blue sclerosis and otosclerosis has been described by Bigleu, Ruttin and others.

5. *Theory of Congenital Origin.*—Alexander held the opinion that the foci of otosclerosis exist even in the fetal period, perhaps like the primordium of all the structures.

Remains of fetal cartilage are occasionally found in the promontory near the anterior margin of the oval window, which, according to some authors, is the predisposing factor for the otosclerotic foci.

Ballenger described in his textbook a vestigial structure in front of the oval window called "fissula ante fenestrum," and the fact that this structure is the site of predilection may favor the theory that it may be the primary focus (Bast). At this stage, it is interesting to note that the osteogenesis of the petrous bone shows essential differences from the ossification of other bones in the human skeleton. As pointed out by Siebenmann, the bony labyrinthine capsule reaches its adult shape and size even in the newborn. The labyrinthine capsule, therefore, always

contains remnants of cartilage and embryonic tissue. The coincidence of portions of fetal structures along with mature bone forms the basis for the genesis of otosclerosis.

6. *Theories Relating to Such Factors as Metabolism and Constitution.*—Some otologists regard disturbances in metabolism as the cause of otosclerosis, and, confirming this view, characteristic biochemical changes have been noted in this condition.

Other conditions, such as anemia, autointoxication, osteomalacia and ozena, have been considered as etiologic factors in otosclerosis.

Toynbee and Buck came forward with a theory that gouty or uric acid diathesis is a contributory factor in otosclerosis. Toynbee, in 1857, described otosclerosis, on the basis of this theory, as stapediovestibular arthritis.

Bauer and Stein made an examination in a large number of cases and found many degenerative signs in patients with otosclerosis. Fisher reported morphologic anomalies in the inner ear of otosclerotic patients, with disturbances of the endochondral ossification and degenerative changes in the inner ear. These findings have been influential in attributing the cause of otosclerosis to a constitutional disposition.

7. *Theory of Syphilitic Origin.*—Habermann stated the belief that otosclerosis, which is essentially an osseous disease of the labyrinthine capsule, is a sequela of acquired or congenital syphilis. But Korner rightly disputed this with the fact that the majority of otosclerotic persons show no positive reactions in serologic studies of the blood.

8. *Theory of Parathyroid Hypofunction.*—This theory was presented by Frey, with the result that parathyroid extracts have been extensively tried, both locally and orally. Some physicians still use them and believe that it helps in reducing the tinnitus. My personal experience with use of this drug in a large number of cases has convinced me of its uselessness.

9. *Theory of Disturbances in Circulation.*—Wittmaack regarded otosclerosis as a result of localized venous stasis in the petrous bone. He stated the argument that local stasis leads to loss of calcium in the bone, halisteresis, which, in turn, leads to resorption of bone. He made the following famous experiment to substantiate his theory: He produced artificial local stasis and congestion in animals by ligating the veins; very similar changes resembling otosclerosis were noted in the bones.

Spurer regarded otosclerosis as a local bone disease due to local alteration in the circulation, as a result of vasomotor imbalance.

Gray expressed the belief that otosclerosis was the result of a defective vasomotor response in the small vessels of the ear.

Recently, in an article, Kobrak [1] attributed the pathologic process of otosclerosis to ischemia. In the article he stated that the well known perivascular otosclerotic osseous changes are regarded as the cause of ischemic lesions in the organ of corti. Otosclerotic changes which involve ischemia in bone are likely to leave the blood supply in the receptoral organ unaltered. One should therefore expect ischemic cochlear degeneration with the earliest stages, and even prestages, of otosclerosis, and one could regard Gray's primary cochlear degeneration as pseudoprimary.

*Personal Theory.*—So far, I have discussed the various important theories I assembled from a fairly thorough study of the various books and literature on the subject. In conclusion, I shall mention my own views on the etiologic factors in otosclerosis. I am inclined to think that Wittmaack's theory of venous congestion is probably the right one. My arguments in favor of the same view are a result of two observa-

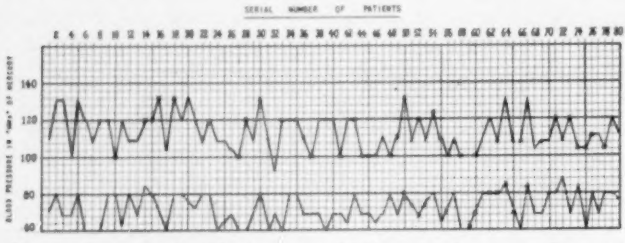


Fig. 2.—Chart showing blood pressure in 80 consecutive cases. The systolic pressure is indicated by the solid line and the diastolic by the broken line. In 70 of the 80 cases the systolic pressure is below 120 and the diastolic below 80 (87.5 per cent); in 46, below 115 and 75 (66 per cent), and in 45 the diastolic pressure was 70 or below, with 70 mm. of mercury (64 per cent).

tions made during my six months stay at the Lempert Institute of Otology, when I had the unique opportunity of studying more than 300 cases, both before and after the fenestration operation for clinical otosclerosis. Those two observations are as follows.

1. The majority of the patients suffering from typical clinical otosclerosis were observed to have low blood pressure.

2. More than two thirds of the patients operated on had the fenestration on their right ear, indicating that the right ear is the ear commonly or first involved, since Lempert, as a rule, operates only on the worse ear.

1. Kobrak, F.: Contributions to Functional Pathology: IV. Clinical Notes on Progressive Congenital Deafness, J. Laryng. & Otol. **61**:49, 1946.

These two observations have made me argue along the following line. It was first noticed by the Japanese anatomist Asahi, and later confirmed by all the anatomists in the world, that the superior sagittal sinus drains into the right transverse sinus in 66 to 70 per cent of human beings. Normally, it is easy for one to imagine the inner ear to be floating, as it were, in a pool of venous blood formed by (1) the transverse sinus; (2) the superior and, especially, the inferior petrosal sinuses; (3) the bulb of the jugular vein, and (4) the cavernous sinus medially. If, on top of these, the superior sagittal sinus drains to one side (the right side), it stands to reason that the right ear may be regarded as drowned in venous blood. In addition to these factors, if the person has a low blood pressure, it will only further augment the venous stasis.

These arguments for the venous stasis theory are confirmed by the findings of Lempert and Wolff, who reported the histologic studies of

*Total Number of Fenestration Operations at the Lempert Institute of Otology for a Six Month Period* *

| | | Per Cent |
|---|---|---|
| Total number of patients operated on | 283 | .. |
| Number of right ears operated on | 264 | 69 |
| Number of left ears operated on | 119 | 31 |
| Number of women patients | 264 | 69 |
| Number of men patients | 119 | 31 |

* The incidence of operation on the right ear is compared to that on the left, and the number of men and women patients are compared. It was purely coincidental that the number of right ears operated on was the same as the number of women patients and the number of left ears operated on was the same as the number of men patients.

the incus and the head of the malleus, routinely removed from 115 ears during fenestration operation, as follows.

1. Distortion in the shape of the ossicles owing to irregular bone contours (57 per cent); more or less extensive fibrotic replacements of bone (23 per cent), and evidence of altered stresses and strains in the ossicular chain

2. Blue mantles

3. Changes in marrow spaces, including tortuosity of vessels; venous stasis; arterial congestion; periarthritis, and fibrosis contributing to generalized fibrotic replacement of bone

4. Pathologic changes in ossicular joints, including erosion or lipping of cartilage; deposition of calcium; overgrowth of atypical bone, and no osteoclasts

Item 3 of this tabulation is an argument in favor of the theory of venous stasis.

Kobrak's theory of ischemia as a causative factor for early otosclerosis does not seem probable. Perhaps it may be argued that in addition to venous stasis anoxemia might be a contributory factor of otosclerosis, which may result from (*a*) venous stasis and (*b*) low blood pressure.

Therefore, on the basis of these two observations, namely, low blood pressure and the greater frequency of involvement of the right ear, in addition to the histologic report of Lempert and Wolff, I am inclined to favor Wittmaack's theory of venous stasis. Further, Wittmaack's famous animal experiment also confirms this supposition.

Hughson and Thompson reported on their various experiments in animals, in which reduction of pressure of cerebrospinal fluid resulted in decrease in hearing response, whereas increase in pressure of the spinal fluid did not show corresponding improvement in hearing acuity. This fact seems to bear out the incidence of low blood pressure in people with otosclerosis.

If the incidence of otosclerosis is more in the right ear to start with, then it will be interesting to find out how the superior sagittal sinus drains in the lesser percentage of cases in which the left ear was operated on, since it was the first to be involved.

Again, if low blood pressure really has an influence on the incidence of otosclerosis then it will be worth while to determine if the progress of the disease can at least be halted by drug therapy, which might increase the blood pressure to normal or slightly above normal, especially if this form of treatment is instituted at a very early stage of the disease.

## INCIDENCE

*Sex.*—Fortunately for men and unfortunately for women, this disease occurs nearly twice as frequently in women as in men. The reason for this still remains unsolved. Is it because of childbearing that women are more frequently affected by the disease?

*Age.*—The disease usually occurs during adolescence, but it is often not noticed, or at least the patient refuses to recognize the disease till hearing drops below the 30 decibel level in at least one ear.

*Distribution.*—Like that of syphilis and tuberculosis, the distribution of otosclerosis is universal and does not seem to have a predilection for any color or race.

## SIGNS, SYMPTOMS AND DIAGNOSIS

The signs and symptoms vary with the site of the lesion. If the usual site of predilection, the oval window, is involved, the typical signs and symptoms of clinical otosclerosis will be present, depending of course, to some extent on whether the stapedial ankylosis is partial or complete.

If, on the other hand, the lesion involves the region of the cochlea, such involvement, of course, being rare, the condition will be diagnosed as nerve deafness, though in reality it will be otosclerosis with clinical nerve deafness.

Again, if the otosclerotic focus involves neither the sound conduction nor the sound perception mechanism but remains within the labyrinthine capsule, the condition will never be diagnosed or suspected, unless the temporal bone is examined at autopsy.

Therefore, Lempert advocated the use of the name "clinical otosclerosis," which will at once signify the suggestive but not the positive evidence of the disease. He presented the argument that the diagnosis of "probable otosclerosis" is necessary from the treatment point of view, but "positive otosclerosis" is possible only by postmortem examination, by methods now available.

*History.*—A family history of hereditary deafness is available in most of the cases and, when it is available, is of great diagnostic value. The lack of such family history may be due to the fact that one of the parents or the grandparents of the patient may have had otosclerosis without the involvement of the region of the oval window and the footplate of the stapes, so that they may not have suffered from "clinical otosclerosis."

*Onset.*—The deafness is characterized by gradual onset, slow progression and by being unilateral in the beginning but always becoming bilateral later. Usually, the onset dates back to adolescence.

*Bezold's Triad.*—This is a very important diagnostic characteristic of the disease. The components of the triad are (1) a markedly negative reaction to the Rinne test; (2) a prolonged reaction to the Schwabach test; and (3) a loss for lower tones.

Deafness for air-borne sound is the most important characteristic, which always begins in the lower frequencies. Bone conduction is prolonged over and above the air conduction level, especially in all those frequencies in which the air conduction level is impaired.

*Tinnitus.*—Patients are frequently annoyed by the presence of continuous or intermittent tinnitus, which may be either mild or severe. Very often patients notice the tinnitus first, and when they see the specialist for it, he discovers their deafness for low tones by air. The tinnitus usually gets worse as the hearing defect progresses, but not infrequently it disappears by itself when the deafness gets severe.

*Paracusis Willisii.*—As the name implies, this condition is a kind of altered hearing acuity, in which the patients notice that they are able to hear better in noisy places. It is perhaps more a comparative than real finding. The various theories to this phenomenon are discussed under a different heading.

*Patency of the Eustachian Tube.*—The eustachian tube on inflation is found to be quite patent, and the hearing level on audiometric examination shows no improvement whatsoever after inflation of the eustachian

tube. Therefore, the very frequent practice of eustachian inflation in cases of otosclerosis should certainly be discontinued.

*Translucency and Atrophic Condition of the Tympanic Membrane.*—The tympanic membrane is usually atrophic and translucent. Even the external canal wall is often thin and widely open. The translucency of the drum membrane often shows the structures of the middle ear rather clearly. The mucous membrane lining the promontory occasionally presents a congested pinkish hue, which has been described by many as pathognomonic of otosclerosis.

*Tuning Fork Tests.*—(*a*) Weber's Test: When there is a pronounced difference in air conduction levels, Weber's test lateralizes the sound of the fork to the worse of the two ears, provided the bone conduction level in that ear is greater than the bone conduction level of the better ear, which is usually the case. But if the bone conduction level is equal in both, the Weber test does not lateralize the sound.

(*b*) Rinne's Test: As mentioned previously, the reaction to this test is markedly negative, as is expected in any conduction type of deafness.

(*c*) Schwabach's Test: The reaction to this test is prolonged to nearly twice that of normal ears provided the cochlea is not affected.

## DIFFERENTIAL DIAGNOSIS

All forms of conduction deafness have to be ruled out in making a diagnosis of otosclerosis. But the most important one which may give rise to difficulty is chronic catarrhal deafness of the middle ear. The possibility of this condition being present is ruled out by the following indications: (1) obstruction or partial obstruction of the eustachian tube; (2) absence of familial history of deafness; (3) absence of stapedial ankylosis, as may be elicited by the Gellé test, and (4) retraction or old scarring of the drum membrane, with thickening.

## TREATMENT

Purely for historical interest, I propose to mention briefly the various methods of treatment tried for otosclerosis, before Lempert's surgical technic came to be recognized by all as the only available useful treatment of the disease clinical otosclerosis.

*A. Nonsurgical Methods.*—(1) Inflation of the Eustachian Tube: It has been the experience of all otologists that in a case of diagnosed clinical otosclerosis the eustachian tube is invariably open. In those very few cases in which difficulty was experienced in blowing in the air, it was due not to blocking of the tube but to improper catheterization as a result of a deflected septum or some such nasal defect. Still, many

otologists have not given up this practice, and I suppose it is done just to satisfy, psychologically, the patient.

(2) Pneumomassage: This is supposed to have some doubtful value in cases in which the bony ankylosis is not well advanced. Politzer

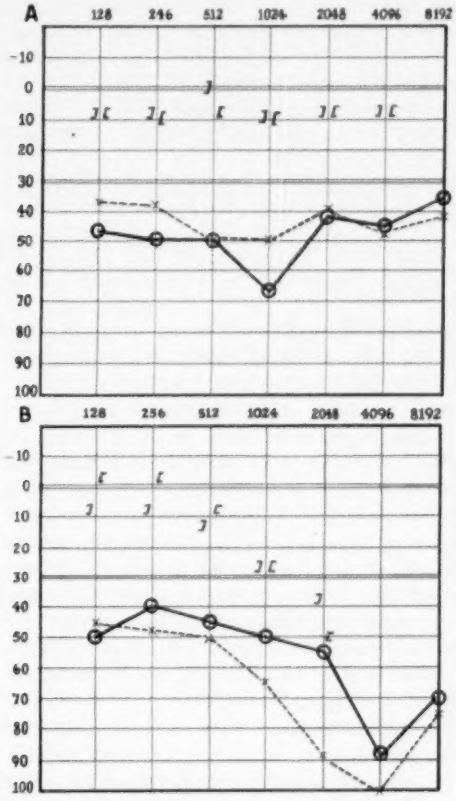


Fig. 3.—Audiograms of 2 patients with otosclerosis; *A*, ideally suitable for fenestration, and *B*, very poorly suitable. The solid line with the circle indicates air conduction for the right ear, the broken line with the cross, for the left. The bracket facing the left indicates bone conduction for the right ear, the bracket to the right, the left ear.

recommended this method as useful in very early stages, at least to halt or delay the progress of the disease. He also recommended the following routine: the pneumomassage is to be done one-half to one minute

each time, two to three times a week, for four to five weeks, followed by rest for several months.

(3) Use of Sodium or Potassium Iodide Orally: Perhaps there is no chronic disease of the human body for which potassium iodide has not been tried. If the physician is at a loss as to the method of treatment, he usually falls back on one of his two good friends, potassium iodide or calcium chloride by the intravenous route. Of course, during the last five to ten years, these two drugs have been displaced by therapy with vitamin B preparation and the sulfanilamides.

Politzer recommended potassium iodide therapy as follows: ½ to 1 drachm (0.03 to 0.06 Gm.) per day, for twenty-five to thirty days, two or three times a year.

(4) Use of Phosporous: Hypophosphate or glycerophosphate were tried with no success.

(5) Use of Parathyroid Extract: This has been tried very extensively, both locally and internally, based on Frey's theory of parathyroid hypofunction.

(6) Use of Vitamin $B_1$ Preparations and Nicotinic Acid: This is one of the recent fads. So far as otosclerosis is concerned, vitamin $B_1$ therapy helps only the manufacturers and not the patients.

*B. Surgical Treatment.*—(1) Extraction of Stapes: Flourens, Kessel and others experimented on birds and rabbits by removing the columella (stapes in birds) or the stapes from the oval window. Deafness ensued, but in some weeks the animal regained normal hearing.

Blake, of Boston, performed the greatest number of these operations on living patients. He perforated the posterior superior quadrant, through which he severed the stapedial tendon and the incudostapedial joint. Then he removed the stapes with a delicate hook, an easy procedure in nonotosclerotic patients, when the stapes was not ankylosed. He reported no benefit with this operation, and his results were confirmed by Knapp, Grunert and Pause at the International Otolaryngological Congress, held in London in 1899.

(2) Fenestration Technic: The main purpose of this technic depends on the establishment of another substitute window to take the place of the oval window, which is closed by the fixation of the footplate of the stapes. The physiologic principle of the operation is that the fluid in the bony labyrinth is incompressible and so requires two openings to set it in motion, or to set waves traveling in its media. The popular view is that sound gets in through one window and gets out by the other, setting up waves in the liquid media. In an otosclerotic ear, the oval window is closed, and it has only one opening, the round window. Normally, the sound is carried by the ossicular chain to the oval window and gets out through the round window. But in a case in which the fenestration has been done, the ossicular chain is destroyed and the sound

waves travel through the membrane of the round window and out through the new window. In other words, the direction of sound travel seems to be reversed.

Holmgren in 1917 first brought forth this operative possibility, and since then he has made a large number of these fistulas of the labyrinth. Most of these fistulas eventually had bony closure, and even before bony closure set in, the hearing improvement was very temporary.

Subsequently, Sourdille reported improvement of long duration, although audiometric tests were lacking and very little confirmatory evidence was available regarding his success.

In 1938 Lempert reported an operation, and since then he has reported many improvements in his technic, with very good and permanent success in many cases. His various steps of polishing and burnishing the fenestra to defy nature's bony regeneration, along with use of the tympanomeatal flap to cover it, has so far produced success in 70 to 80 per cent of cases. One can be sure that before long he will perfect the technic so well that success with a permanent cure will be attained in at least 95 per cent of cases.

For a detailed study of Lempert's technic, the reader is referred to Lempert's various reports and textbooks.

## COMMENT

Before concluding this paper, I wish to present some of the interesting aspects in the study of otosclerosis noted by Lempert in his institute.

1. *Pinkish-Hued Promontory.*—This has been described in many textbooks as frequently occurring pathognomonic evidence of otosclerosis. But it has been observed to be but rarely present in the patients examined by Lempert. When this condition was present and when the progress was found to be rapid, Lempert called such cases "fulminating types of otosclerosis." When patients with the fulminating type of otosclerosis, evidenced by the pinkish promontory, were operated on, the labyrinth was invariably found to be hyperemic. In such patients the cochlea seemed to be involved early, and, accordingly, they were not suitable for the fenestration operation, as was evident from their postoperative progress.

2. *Paracusis Willisii.*—As mentioned previously, this is a condition in which the otosclerotic patient seems to hear better in noisy places. There are three theories to explain this phenomenon. The first theory is Politzer's, who attributed it to "shaking up of the ossicles," whose joints have become rigid. In support of this view, it has been observed that when a deep-vibrating tuning fork is placed on the cranium, hearing is improved. Similar improvement in hearing was noted even when a toneless, rapidly vibrating body was placed on the cranium. Further, these experiments did not improve the hearing in patients with nerve deafness.

The second theory is that of Lowenburg and Urbantschitsch, who attributed the condition to increased irritability of the acoustic nerve brought about by concussion.

The third view is that of Lempert. He stated that the stapes in normal persons gets jammed in noisy places as a mechanism protective of the eighth nerve, but in otosclerotic patients with partial ankylosis, this protective mechanism is unable to function, owing to fixation of the stapes; therefore these persons seem to hear comparatively better than or as well as normal persons.

3. *Prognosis.*—It has been noted by Lempert that if there are 2 patients with otosclerosis, with identical findings on audiometric, fork, examination, etc., and if one is more than 50 years and the other between 20 and 30, the prognosis is better in the older person. This paradoxical finding is explained by the fact that the chance of bony regeneration is much greater in the younger patient than in the elderly one, in whom the growth factor is comparatively small.

4. *Bony Regeneration.*—By routine revision of cases when the hearing went down after six months or more of good improvement, Lempert noticed that in most cases the bony regeneration was from the endosteal layer. The endosteum is developed from the cartilaginous capsule of the otic vesicle of the embryo, whereas the periosteum is derived from the mucoperiosteum of the middle ear. It may be that the developmental origin has some bearing on the different rates of bony regeneration. There may also be another explanation for the lack of regenerative power of the periosteal layer of the fenestra nov-ovalis: It may be due simply to the fact that all the polishing and burnishing technics of Lempert are applied more to the periosteal layer than to the endosteal layer, which, by virtue of its inner position, escapes these technics. Therefore, it is obvious that the haversian system, blood supply, etc., of the periosteum are completely destroyed, preventing its regeneration; whereas those of the endosteum are intact, thus enabling it to regenerate easily.

5. *Tinnitus.*—When patients that have both otosclerosis and tinnitus are operated on for otosclerosis by fenestration, they have a marked decrease in the tinnitus as hearing improves. Very often the tinnitus stops completely when the hearing level goes up above the 30 decibel level.

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Dr. Julius Lempert furnished various data for use in this thesis and permitted me to use certain of his observations.

# THROMBOSIS OF THE CAVERNOUS SINUS

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WITH the advent of the sulfonamides and, later, of penicillin and streptomycin, thrombosis of the cavernous sinus has passed from the list of unsolvable otolaryngologic problems. This condition has been considered an otolaryngologic problem for the reason that the causes of thrombosis of the cavernous sinus are chiefly diseases of the nose and ear and therefore come within the dominion of the otorhinologist.

The first case was found at autopsy and reported by Duncan in 1821.[2] The first clinical report of the condition was made by Vigla in 1839.[3] Bircher [4] in 1893 reported the first case with recovery following surgical procedure. Grove [5] in 1936, after reviewing all the cases reported up to that date, stated that in the acute fulminating type of thrombosis or thrombophlebitis of the cavernous sinus the mortality rate was almost 100 per cent and that in all the other types the rate was 93 per cent. In 1943, MacNeal, Frisbee and Blevins,[6] in bringing the subject up to date, could find only 17 cases reported with recovery from all forms of treatment in the acute fulminating type. Since then, more than 90 cases with recovery have been reported; of these, 17 were the acute fulminating variety, in which Staphylococcus aureus was obtained on culture.

Although there have been a large number of recent reports of recovery, I believe this case is worthy of reporting for the two following

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Read before the Section on Otolaryngology of the College of Physicians of Philadelphia, April 28, 1949.

1. Footnote deleted.

2. Duncan, cited by Chisolm, J. J., and Watkins, S. S.: Twelve Cases of Thrombosis of the Cavernous Sinus, Arch. Surg. **1**:483 (Nov.) 1920.

3. Vigla, cited by Dixon, O. J.: The Pathologic Examination of Cavernous Sinus Thrombosis as Guide to Diagnosis, Prognosis and Treatment, J. A. M. A. **87**:1088 (Oct. 2) 1926.

4. Bircher, H.: Phlebitis des Sinus transversus, petrosus und cavernosus: Eröffnung und Disinfektion nach Entfernung der Felsenbeinpyramide—Heilung, Centralbl. f. Chir. **20**:483, 1893.

5. Grove, W. E.: Septic and Aseptic Types of Thrombosis of the Cavernous Sinus: Report of Cases, Arch. Otolaryng. **24**:29 (July) 1936.

6. MacNeal, W. J.; Frisbee, F. C., and Blevins, A.: Thrombophlebitis of the Cavernous Sinus: A Review of Reported Recoveries with Special Reference to Thrombosis of Staphylococcic Origin, Arch. Ophth. **29**:231 (Feb.) 1943.

reasons: It is the first case to be reported in the literature in which streptomycin was used, and it is the only case in which evidence was presented of intracranial bleeding resulting from treatment with heparin.

The anatomy of the cavernous sinus has been fully covered by Eagleton,[7] Campbell,[8] Grove,[5] MacNeal and associates[6] and Goufas,[9] and therefore will not be discussed here. Types of infection and thrombus have been adequately covered in previous papers.

The diagnostic criteria set down by Eagleton[7] will, in all probability, have to be modified somewhat, since early and intensive treatment with penicillin often nullifies one or more of his stipulations. However, in the case reported here, all six criteria described by Eagleton were present.

The first great change in treatment came in 1939 with the use of sulfanilamide, mentioned in case reports by Barnshaw,[10] Seydell[11] and Cadbury.[12] The use of sulfanilamide was followed by that of other chemotherapeutic agents, with a noticeable decrease in mortality and morbidity.

At approximately the same time as the sulfonamides were introduced, MacNeal and associates[13] introduced the use of bacteriophage, which lowered the mortality rate from approximately 100 per cent, as stated by Grove,[5] to 43 per cent. However, owing to the technical difficulties and the prolonged treatment required, this method was rapidly replaced by chemotherapy.

The next great advance, the use of penicillin, was reported by Goodhill[14] in 1944. Since then, more than 40 cases of recovery with use of this drug have been recorded in the literature.

As the causative organisms develop resistance to the sulfonamides and penicillin, other antibiotic drugs, such as streptomycin and aureo-

7. Eagleton, W. P.: Cavernous Sinus Thrombophlebitis, New York, The Macmillan Company, 1926.

8. Campbell, E. H.: The Cavernous Sinus: Anatomical and Clinical Considerations, Ann. Otol., Rhin. & Laryng. **42**:51, 1943.

9. Goufas, G.: Contribution a l'étude générale de la thrombophlébitis du sinus caverneux o'origine antérieure, Rev. de laryng. **68**:168, 1947.

10. Barnshaw, H. P.: Report of a Case of Bilateral Cavernous Sinus Thrombosis: Recovery with Operative Intervention, J. M. Soc. New Jersey **36**:22, 1939.

11. Seydell, E. M.: Recovery from Thrombosis of the Cavernous Sinus, Arch. Otolaryng. **30**:429 (Sept.) 1939.

12. Cadbury, W. W.: Cavernous Sinus Thrombosis: First Cure by Sulfonamide Drugs (Prontosil and Sulfanilamide), Chinese M. J. **65**:91, 1947.

13. MacNeal, W. J.; Frisbee, F. C., and Blevins, A.: Bacteriophage Therapy of Staphylococcic Septic Obstruction of the Cavernous Sinus: Report of Cases, Arch. Ophth. **29**:341 (March) 1943.

14. Goodhill, V.: Penicillin Treatment of Cavernous Sinus Thrombosis, J. A. M. A. **125**:28 (May 6) 1944.

mycin, will be added to the therapeutic armamentarium used for this still serious and not uncommon condition.

In 1941, another drug, heparin, was introduced and reported on by Lyons,[15] and, later that year, by Schall.[16] Bancroft [17] in 1940 speculated on its use: "For the septic thrombosis occurring in association with otolaryngologic conditions it would seem advisable to combine chemotherapy with heparin. . . . The combination of the sulfanilamide group and heparin should aid in prevention of spread of septic emboli and in stabilization of the clot when it is impossible to ligate the vein proximally." Lyons,[15] in reporting his 2 cases in 1941, made the following statement: "There is reason to believe that the staphylococcal bacteriemia usually arises from foci of septic thrombophlebitis in and around areas of suppuration. Such a conception indicates that the attack on any staphylococcal bacteriemia should attempt to prevent the extension of septic thrombi as well as to destroy the bacteria." Both Lyons and Schall used heparin in a continous drip, keeping the clotting time of the patient's blood at approximately ninety minutes. Their 3 patients recovered without serious side effects. In opposition to this theory, Rigdon and Haynes [18] in 1942 reported that in their experiments heparin in a 25 per cent concentration did not inhibit the clotting of blood infected by the "coagulase" strains of staphylococci.

In 1941, Ershler and Blaisdell,[19] using larger doses of heparin in conjunction with sulfathiazole, reported a case in which uncontrollable hematuria developed and led to a dangerous anemia. When the number of red cells reached 2,520,000, use of heparin was stopped, and recovery began with the use of sulfadiazine and blood transfusions.

Other authors have also advised against the use of heparin. Moore, Gardner, Bell, and Tannenbaum [20] in 1942 made the following statement with relation to this: ". . . having seen the brains which Mallory demonstrated at the Massachusetts General Hospital, showing the vast cerebral hemorrhages following the administration of heparin, . . . we proceeded as we had been, placing all confidence in chemotherapy."

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15. Lyons, C.: The Treatment of Staphylococcal Cavernous Sinus Thrombus with Heparin and Chemotherapy, Ann. Surg. **113**:113, 1941.

16. Schall, L. A.: Treatment of Septic Thrombophlebitis of the Cavernous Sinus, J. A. M. A. **117**:581 (Aug. 23) 1941.

17. Bancroft, F. W.: Use of Anticoagulants in Cases of Post-Operative Thrombosis and Embolism, Arch. Otolaryng. **32**:934 (Nov.) 1940.

18. Rigdon, R. H., and Haynes, A.: Observation on the Failure of Heparin to Inhibit the Clotting of Blood In Vitro by Staphylococci, Ann. Surg. **116**:430, 1942.

19. Ershler, I. L., and Blaisdell, I. H.: Massive Hematuria Following Use of Heparin in Cavernous Sinus Thrombosis, J. A. M. A. **117**:927 (Sept. 13) 1941.

20. Moore, G. B., Jr.; Gardner, J. D.; Bell, K. R., and Tannenbaum, A. J.: Cavernous Sinus Thrombophlebitis: Cure with Sulfathiazole, J. A. M. A. **119**:708 (June 27) 1942.

Their patient recovered with the use of sulfadiazine. Kelen,[21] in reporting the recovery of a case with the combination of penicillin and sulfathiazole in 1947, considered heparin an unnecessary and dangerous adjunct.

However, heparin was used by Wiesenfeld and Phillips[22] in 1944; Muntarbhorn[23] in 1945; Henner and Ridall[24] in 1945; Welty[25] in 1946; Reich, Likely, Yahr and Baron[26] in 1946 (they switched to dicumarol® after the second day); Stool and Lomas[27] in 1948 (who also used dicumarol® after the second day); Bucky, Lahey and Kunkel[28] in 1948, and, possibly, others. Fox and West[29] in 1947 used dicumarol® as the only anticoagulant.

For the treatment of thrombophlebitis in general, deTakats[30] stated the opinion that heparin was equally effective and that treatment with it was simplified by administration of 50 mg. intravenously every four hours, with the coagulation time determined hourly from the first period in order to find the patient's tolerance to the drug. Both he and Moore[31] stated that heparin and dicumarol® are safe, if carefully administered, and that dicumarol® is easier to use and cheaper but more dangerous because of its accumulative and prolonged effect and its delayed action. Moore made the following statement: "I have become convinced that dicumarol® must never be used without frequent pro-

21. Kelen, A.: A Case of Cavernous Sinus Thrombosis with Recovery, Canad. M. A. J. **57**:578, 1947.

22. Wiesenfeld, I. H., and Phillips, E.: Thrombophlebitis of a Cavernous Sinus Following Extraction of Teeth: Cure with Penicillin and Heparin, Arch. Otolaryng. **40**:497 (Dec.) 1944.

23. Muntarbhorn, S.: Cavernous Sinus Thrombosis Treated with Heparin and Sulfathiazole, Lancet **1**:10, 1945.

24. Henner, R., and Ridall, E. G.: Thrombosis of the Cavernous Sinus: Treatment with Penicillin and Heparin, Arch. Otolaryng. **41**:296 (April) 1945.

25. Welty, R. F.: Bacterial Thrombophlebitis of a Cavernous Sinus with Recovery, Arch. Otolaryng. **43**:70 (Jan.) 1946.

26. Reich, C.; Likely, D.; Yahr, M., and Baron, R.: Case of Cavernous Sinus Thrombophlebitis Successfully Treated by Combined Anticoagulants and Chemotherapy, Ann. Int. Med. **24**:1093, 1946.

27. Stool, J. A., and Lomas, R. D.: Thrombophlebitis of the Cavernous Sinus with Recovery, Texas State J. Med. **44**:372, 1948.

28. Bucky, T. L.; Lahey, W. J., and Kunkel, P.: Bilateral Cavernous Sinus Thrombophlebitis: Complete Recovery with Multiple Systemic Complications, Connecticut M. J. **12**:996, 1948.

29. Fox, S. L., and West, G. B., Jr.: Thrombosis of Cavernous Sinus: Newer Concepts in Its Management and Report of Three Cases, J. A. M. A. **134**:1452 (Aug. 23) 1947.

30. deTakats, G., in Christopher, F.: Textbook of Surgery, ed. 4, Philadelphia, W. B. Saunders Company, 1945.

31. Moore, R. M.: Blood Coagulation and Its Clinical Modification with Anticoagulants, Texas Rep. Biol. & Med. **5**:221, 1947.

thrombin time checks, preferably daily checks and necessarily so until the maintenance dose has been carefully established in the individual patient. Furthermore, the day's dose of dicumarol® must never be ordered until that day's prothrombin time has been reported."

From the opinions reviewed, together with that expressed in the report of Ershler and Blaisdell,[10] as well as from the material gleaned in this and from the rarity of reports of recovery without side effects in cases in which anticoagulants were used, I have come to the conclusion that such drugs should be used only with the utmost caution and that their addition to the accepted regimen of treatment should await further study.

## REPORT OF A CASE

L. H. G., a Negro aged 40, was first seen on Nov. 7, 1948. He had been in good health until November 1948, when he noticed a small "pimple" on his right cheek. He opened it, but when it began to swell and become tender, he called on his local doctor, who incised the lesion and gave him an injection of penicillin. The lesion continued to enlarge, the face became swollen and the right eye swelled shut. He again consulted his local doctor, who gave him another injection of penicillin. But when, on the following day, his left eye also began to swell, the patient came to the receiving ward of the Pennsylvania Hospital in Philadelphia. He was admitted immediately to the otolaryngologic service.

Physical examination showed a well developed Negro aged 40 in acute distress. He was mentally alert and cooperative. There was a firm swelling extending over the entire right side of the face. A discharging lesion, 2 mm. in diameter, was seen 1.5 cm. lateral to the right corner of the mouth. The right facial and angular veins could be palpated as far as the inner canthus. The right eye was shut with a second degree proptosis, marked chemosis, orbital edema and ptosis of the right lid. The pupil of the right eye was fixed and dilated; there was paralysis of the right lateral rectus muscle, and a creamy discharge at the canthus. The left eye was open without evidence of nerve involvement, but there was a moderate amount of orbital edema. Funduscopic examination of both eyes showed no pronounced venous stasis. The right nostril was swollen closed, and a small amount of dried blood was seen along the nasal floor.

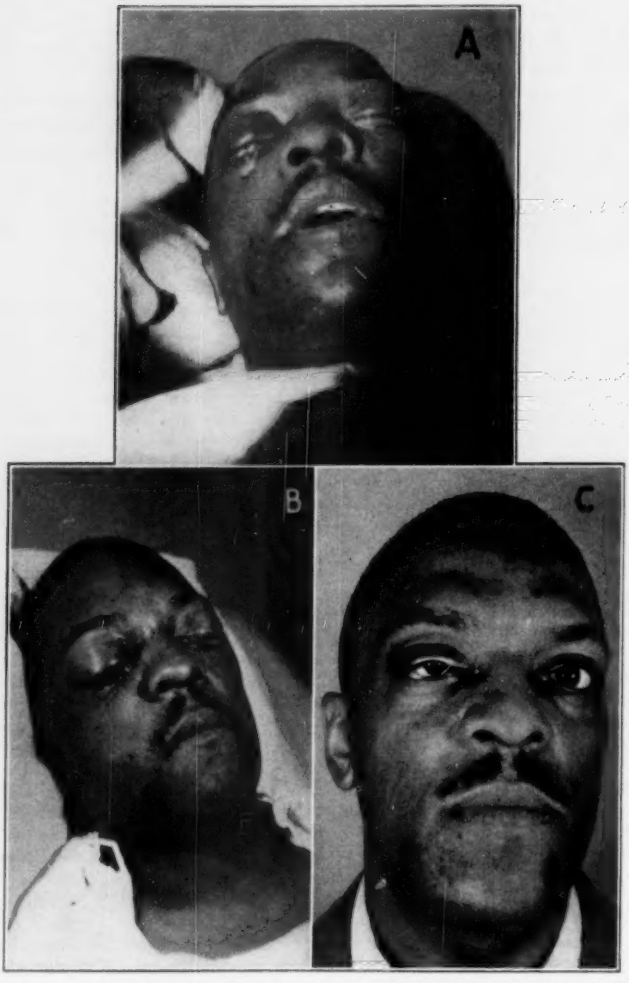
The temperature was 102 F. The white cell count was 33,000; the red cell count was 4,900,000, and the hemoglobin was 16 Gm. The urine gave a 2 plus reaction for albumin, but was otherwise normal. Spinal puncture showed a spinal pressure of 230 mm. of water with 540 white cells and 55 per cent polymorphonuclear leukocytes. The physical examination otherwise indicated nothing abnormal.

Cultures were made of the blood and spinal fluid, from the lesion on the cheek and from the inner canthus.

The patient was placed on treatment with 100,000 units of penicillin every three hours; 4 Gm. of sulfadiazine was given immediately and 1 Gm. was given every three hours. Intravenously, 50 mg. of heparin was administered every four hours.

Three hours after the patient's admission to the hospital, the edema and chemosis had increased in the left eye, and paralysis of the left external rectus muscle was noted.

The cardiovascular service was consulted concerning the use of anticoagulants. The members advised that heparin be used instead of dicumarol® and that the




Photographs (*A*) taken on second day of hospitalization; the initial lesion can be seen as a dark area on the right cheek, lateral to the angle of the mouth; (*B*) taken on Nov. 10, 1948, as patient began to improve; (*C*) taken twenty days after patient's discharge from hospital. Since then, the ptosis of the right lid has disappeared.

coagulation time be kept at or near thirty minutes, stating that intermittent doses of 50 mg. every four hours would be adequate. The ophthalmologic and neurosurgical departments were called in consultation, and the members suggested the use of streptomycin, which was started at 2 Gm. a day.

The patient's condition remained unchanged for the first three days; his temperature rose to 103 F., and both eyes were swollen shut.

On November 10 reports indicated that the cultures from the facial lesion and the lesion in the right eye yielded hemolytic Staphylococcus aureus, which was coagulase positive, not susceptible to sulfadiazine but moderately susceptible to penicillin and streptomycin.

The patient began to improve that same day, with a slight decrease in the orbital edema. The temperature was down to 102 F. Both lids were affected with ptosis, and both lateral recti and the superior oblique muscles were paralyzed. There was congestion of the orbital veins but no complete blockage.

After the 2 a. m. injection of heparin on November 10, the patient became disoriented and had a severe chill. The 6 a. m. injection was omitted, and the coagulation time was found to be 35 minutes.

A blood culture taken on his entry to the hospital was reported positive for Staph. aureus on the next day. The culture of the spinal fluid was free from organisms.

The patient continued to improve, with function of the left eye gradually returning. The temperature was down to 100 F. on November 12 when, following the 6 a. m. injection of heparin, he became very apprehensive and disoriented for approximately one hour and, subsequently, completely comatose, with a spastic paralysis of the right arm. The left eye was wide open. Both pupils responded to light and the fundi were clear. The neck was stiff, and the patient did not respond to painful stimuli. The deep reflexes were hyperactive, but Babinski's sign was not elicited. The coagulation time was 40 minutes. Spinal puncture showed a pressure of 235 mm. of water; the fluid was xanthochromic and contained 2,160 red cells and 33 white cells. The heparin was immediately counteracted with protamine zinc insulin. This condition persisted for the remainder of the day until approximately 4:30 p. m., when consciousness returned gradually. The patient was very apprehensive, depressed and nervous. A generalized weakness persisted in all extremities for the next twelve hours.

I felt that generalized cerebral bleeding had occurred as a result of the decrease in coagulation time and accordingly discontinued the heparin.

After the discontinuance, the patient improved steadily. Streptomycin was discontinued on the thirteenth day, the temperature having been normal for three days. On the same day, the treatment with penicillin was changed to that of a daily dose of 300,000 units of duracillin,* which was stopped on the twentieth day. The chemosis had completely subsided in the left eye and was greatly decreased in the right on November 26. The bilateral paralyses of the fourth and sixth nerves persisted.

The patient was discharged on November 30, the twenty-fourth day of hospitalization. On discharge, there was a slight ptosis and proptosis of the right eye and bilateral paralysis of the fourth and sixth nerves, with returning function of the left eye.

He was followed in the outpatient department, and when he was last seen on Nov. 14, 1949, there was complete restoration of normal function of the extraocular muscles bilaterally.

SUMMARY

A brief discussion is given on thombosis of the cavernous sinus, with the latest treatment and particular reference to the use of anticoagulants.

A case of thrombophlebitis of the cavernous sinus is presented, in which recovery was attained with the use of penicillin, streptomycin and heparin. On the fourth day of use of heparin, the patient showed evidence of cerebral bleeding, and the heparin was discontinued.

122 East Sixty-Fifth Street.

# Case Reports

## TUMOR OF THE GLOMUS JUGULARIS

D. A. DE LISA, M.D.
SCHENECTADY, N. Y.

Tumors of the glomus jugularis have only recently been recognized, and few cases have been reported. A brief review of the literature, summary of the clinical characteristics and the presentation of an additional case are the purpose of this report.

### REVIEW OF LITERATURE

Guild [1] in 1941 reported his discovery in the temporal bone of a minute structure, which he termed glomus jugularis. It is histologically identical with the carotid body and is situated in the adventitia of the dome of the jugular bulb covered by the bone of the hypotympanum. Its innervation and blood supply are the glossopharyngeal nerve and ascending pharyngeal artery. In this respect also it is similar to the carotid body, which has the same blood and nerve supply.

Rosenwasser [2] in 1945 made the first report of a tumor of the glomus jugularis. Additional cases have since been reported by Kipkie,[3] Le Compte and associates,[4] Winship and associates [5] and Lundgren.[6]

Winship reported 2 cases and made a comprehensive survey of tumors of the tympanic region which had been reported in the literature as hemangioendothelioma, endothelioma, etc. He was able to obtain histologic sections of several of these and proved them to be glomus jugularis tumors. He expressed the belief that many others of the more than 50 reported cases of vascular tumors of this region are probably glomus jugularis tumors.

Lundgren reported 4 cases and also reviewed cases reported in the literature as hemangioendothelioma, etc. Apparently unaware of the published report by Winship and associates, he arrived at similar conclusions regarding their true nature. He stated the opinion that they should be called tympanic body tumors rather than glomus jugularis tumors.

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From the Department of Otolaryngology, Albany Medical College and Albany Hospital.

1. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus-Jugularis, in Man, abstracted, Anat. Rec. (supp. 2) **79**:28, 1941.

2. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid, Arch. Otolaryng. **41**:64-67 (Jan.) 1945.

3. Kipkie, G. F.: Simultaneous Chromaffin Tumors of the Carotid Body and the Glomus Jugularis, Arch. Path. **44**:113-118 (Aug.) 1947.

4. Le Compte, P. M.; Sommers, S. C., and Lathrop, F. D.: Tumor of Carotid Body Type Arising in the Middle Ear, Arch. Path. **44**:78-81 (July) 1947.

5. Winship, T.; Klopp, E. T., and Jenkins, W. H.: Glomus-Jugularis Tumors, Cancer **1**:441-447, 1948.

6. Lundgren, N.: Tympanic Body Tumors in the Middle Ear, Acta otolaryng. **37**:367-379, 1949.

### CLINICAL CHARACTERISTICS

The tumor is three times as common in women as in men and is usually found in patients over 40 but does occur in children. It is usually benign but locally invasive. Winship reported only 1 case with metastases to the cervical nodes. Except in children, its course is very slow, with a usual history of many years' duration.

The commonest signs and symptoms are deafness and tinnitus, an aural polyp which bleeds profusely when traumatized, otorrhea and facial paralysis.

It causes damage by destroying adjacent structures in its slow expansion. Surrounding bone undergoes pressure necrosis. The facial nerve is commonly affected. Other cranial nerves, especially the ninth, tenth and eleventh within the jugular foramen, may be involved. It sometimes invades the petrous pyramid but usually grows outward into the external canal. It is apparently radioresistant, and complete removal is difficult.

For a more thorough discussion reference may be made to the excellent reports by Winship [5] and Lundgren.[6]

### REPORT OF A CASE

*History.*—A white woman aged 64 was first seen by the late Dr. Harry K. Tebbutt Jr. on Jan. 24, 1949. Approximately in 1944 she had first noticed tinnitus and deafness in the right ear. The tinnitus was described as a constant low-pitched hum. Sometime in 1947 she began to have pain in the right ear and sought medical help. She was told she had a tumor in the ear. She was hospitalized and the tumor removed without undue bleeding or other complications. The tinnitus and deafness persisted but the pain was relieved. In 1948 she was told that the tumor had recurred. She was again hospitalized, and secondary removal was performed. Following this the ear "drained" for several weeks, but there was no bleeding. Four months later she was told the tumor had again recurred and she was referred to Dr. Tebbutt. The right external auditory canal was filled with a firm, grayish mass, and removal was advised. On Jan. 26, 1949, removal was performed in the outpatient department and was followed by profuse bleeding. Packing and hospitalization were required. The following day the packing was removed without bleeding, and she was discharged. For several weeks thereafter there was discharge from the ear but no pain or bleeding. The tinnitus and deafness persisted.

*Examination.*—The patient first came under my care on July 1, 1949. She was in good health and had no complaints except for tinnitus and deafness of the right ear. The right external auditory canal was dry. The region usually occupied by the tympanic membrane consisted of smooth, irregular, dry, glistening, beefy red, firm tissue. There was no pulsation or discharge, and the canal was normal in shape and almost normal in depth. There were no scars to indicate that anything but local removal had been done. Tuning fork tests revealed a severe degree of conduction deafness of the right ear. A reliable audiogram was not obtained because of inconsistent responses by the patient. Examination otherwise gave results within normal limits.

*Laboratory Data.*—A hemogram was within normal limits. The blood reaction to the Wassermann test was negative. Roentgenographic films of the mastoid process revealed "clouding" in the region of the antrum on the right but otherwise indicated nothing abnormal. The pathologic report of the specimen removed on January 26 indicated "glomus jugularis tumor." Examination and report were made by

Dr. Arthur W. Wright (chief pathologist Albany Hospital) in consultation with Dr. Fred W. Stewart (attending pathologist, Memorial Hospital, New York City). The histologic appearance resembled that of a tumor of the carotid body.

*Examination.*—When last seen on October 1 the ear was still dry and painless. The only symptoms were tinnitus and deafness of the right ear. There was no change in appearance of the lesion. The probability of local extension was explained to the patient. She refused radical treatment but agreed to remain under observation.

SUMMARY

A case of glomus jugularis tumor of the right ear in a 64 year old white woman is reported. Clinically and histologically it is similar to other cases reported in the recent literature. Recognition of these tumors was made possible by the discovery of Guild in 1941 of the normal glomus jugularis. These tumors were previously reported usually as hemangioendothelioma. Thorough discussions of these tumors have been published by Winship and his associates [5] and Lundgren.[6]

1408 Union Street.

# Clinical Notes; New Instruments and Technics

## EAR ASPIRATOR AND INJECTOR

BARNARD C. TROWBRIDGE, M.D., D.Sc.(Med.)
KANSAS CITY, MO.

Owing to recent interest shown in the aspiration and injection method of treating conditions of the middle ear, an instrument has been devised to simplify these procedures.

The aspiration of fluids from the middle ear cavity, such as in cases of otitis media with effusion,[1] formerly required the help of an assistant during aspiration, since the operator steadied the syringe and needle with one hand while holding the electric otoscope with the other. The assistant withdrew the syringe plunger

*A*, ear aspirator and injector; *B*, ear aspirator assembled for use.

to aspirate the tympanic fluid. This act is often difficult to accomplish without movement of the needle in the tympanic membrane, which should be avoided.

The unassembled syringe holder for aspiration and injection of the tympanum is shown in part *A* of the figure.[2]

The syringe is securely fixed in the holder by a set-screw. A recoil spring is placed over the barrel of the syringe plunger, which is depressed prior to piercing the drum head. Release of the thumb on the plunger permits the recoil of the spring, resulting in aspiration of the tympanic fluid. In this manner, the entire procedure is accomplished without assistance (figure, *B*).

From the Department of Otolaryngology of the University of Kansas Medical Center, Kansas City, Kan.

1. Trowbridge, B. C.: A New Treatment of Acute Aero-Otitis Media, Arch. Otolaryng. **50**:255 (Sept.) 1949.

2. The ear aspirator and injector is made by George Pilling & Sons, of Philadelphia.

Injection into the tympanum [3] of ethylmorphine hydrochloride [4] is simplified by the syringe holder, since manipulation of the syringe with the attached needle is performed with ease when it is securely fixed in the holder.

194 Plaza Time Building.

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3. Goldstein, M. A.: Thyroxine in the Treatment of Otosclerosis: Preliminary Report, Laryngoscope **46**:112 (Feb.) 1936; Thyroxine Therapy in Otosclerosis: Report of 42 Cases, ibid. **48**:443 (July) 1938. Trowbridge, B. C.: Injection of the Tympanum for Chronic Conductive Deafness and Associated Tinnitus Aurium: A Preliminary Report on the Use of Ethylmorphine Hydrochloride, Arch. Otolaryng. **39**:523 (June) 1944; Tympanosympathetic Anesthesia for Tinnitus Aurium and Secondary Otalgia, ibid. **50**:200 (Aug.) 1949.

4. The ethylmorphine hydrochloride solution for tympanic injection and the cocaine and aniline oil solution for topical anesthesia are prepared by the C. Morris Watkins Drug Company, of Kansas City, Mo.

# Abstracts from Current Literature

## Ear

Relative Auditory Aphasia. M. Saltzman and M. S. Ersner, Eye, Ear, Nose & Throat Monthly **28**:523 (Nov.) 1949.

The term "relative auditory aphasia" is used by the authors to designate the form of speech deafness which becomes superimposed on a severe hearing impairment as the result of neurologic and psychophysiologic factors. In profound deafness, the understanding of speech frequently deteriorates to a greater degree than the pure tone acuity. This is particularly true in people of advanced age. Another important factor in these patients is the psychogenic overlay, which is most frequently present. The patient ceases to pay attention to the spoken word when he has to exert too much effort to hear. Auditory training is of value for these patients.

Jennes, Waterbury, Conn.

The Hard of Hearing Program in the Public Schools of Pennsylvania. James E. Landis, Pennsylvania M. J. **52**:1380 (Sept.) 1949.

The Committee on the Conservation of Hearing for the state of Pennsylvania reports that there are approximately 45,000 school children in the state with various degrees of deafness. This means that from 1 to 3 children in every schoolroom have trouble with their hearing, or from 3 to 4 per cent of the school children in the state.

Hearing tests alone do not constitute an adequate conservation of hearing program. The tests reveal the identity of the children in need of special medical and educational attention. Hearing programs must have the cooperation of local physicians, audiometric technicians and local school personnel.

The public must be educated to the fact that neglect and improper care will create a handicap in the pupil, both from an educational and from a psychologic standpoint.

The State Legislature through the Department of Public Health has approved medical examinations by a certified otologist on children showing a loss of 20 or more decibels in two or more frequencies, and it is hoped to have all of the cases classified after two years of examinations. This is a decided step forward in determining the cause of early deafness.

Author's Abstract, Reading, Pa.

Endaural Surgery of the Temporal Bone. C. A. Swanson, Laryngoscope **59**:984 (Sept.) 1949.

Swanson reviews the history of endaural temporal bone surgery and points out the advantages of that method of approach. The first 25 patients with otosclerosis who were operated on at the Bethesda Naval Hospital are discussed, and each case is presented separately. Sixteen of the 25 had a return of useful serviceable hearing. Mistakes incurred in the series are emphasized with the thought in mind of encouraging and helping others who are beginners in this field.

Hitschler, Philadelphia.

## Larynx

DELAYED PNEUMONIA AND URTICARIA FOLLOWING BRONCHOGRAPHY. H. E. BASS, New England J. Med. **240**:505 (March 31) 1949.

The author reports a case of delayed pneumonia and urticaria following bronchography in an allergic person and recalls that previously similar cases were believed to be due to an allergic reaction to the iodide component of iodized oil U. S. P. (lipiodol®).

More recently, 3 cases of pneumonia following the use of iodized oil in asthmatic patients were described by Cooperstein that differed from those previously reported. A delayed onset of pneumonia was noted, varying between nine and twelve days.

In addition, these cases were characterized by certain allergic manifestations, such as urticaria, blood eosinophilia and positive reaction to sensitivity tests with iodide-containing compounds. The delayed pneumonic reaction was described as resembling the delayed reaction of serum sickness.

This lag was related to that in the present case report of a 59 year old man who was admitted to Municipal Sanatorium, Otisville, N. Y., on July 17, 1947. At this time it was suspected that he had pulmonary tuberculosis. A bronchoscopy was performed, with no abnormal findings. On July 31, a bronchogram was taken. About 8 cc. of iodized oil was instilled into both lobes of the left lung by means of an intratracheal catheter. The bronchogram showed suggestive bronchiectatic changes in the field of the left upper lung. Fourteen days later the patient first noted the onset of productive cough. The temperature, which had been normal, rose to 100.4 F. At the same time, an extensive itching cutaneous eruption, which was urticarial and covered the arms, legs and trunk, developed. The temperature fell by lysis and reached normal levels after five days. A roentgenogram of the chest taken on September 3 showed some regression of the area of pneumonic density, which still occupied the base of the lung. No allergic studies were performed at the time of the acute illness. However, a blood count done two weeks after the disappearance of the urticarial eruption did not reveal any increase in eosinophilic leukocytes.

Bass emphasizes that because of the possibility of late reaction to injection with iodized oil the patient should be kept under observation for at least two weeks after bronchography and that iodide sensitivity tests should be performed on patients who have familial or personal history of allergy.

GOODALE, Boston.

## Nose

THE TREATMENT OF SINUSITIS IN CHILDREN. VICTOR GOODHILL, California Med. **70**:177 (March) 1949.

The sulfonamides and antibiotics have been of great value in reducing the duration and severity of acute sinusitis in children. Chemotherapy in the acute disease will probably prevent much chronic sinusitis of the infectious type. The commonest variety of chronic sinus disease is caused by a primary allergic condition plus secondary infection. It is impossible to treat these conditions successfully without treating the allergy as well as the infection. The home use of any nose drop preparation is of little value in the treatment of chronic sinusitis of any type or localization.

The local nasal use of sulfonamides or antibiotics is not based on rational principles. Their clinical value is negligible. They may, moreover, be decidedly

irritating to the nasal mucosa. One should not hesitate to resort to rational surgical procedures to improve nasal ventilation in a child with sinusitis. While the advent of chemotherapeutic, antibiotic and antihistaminic drugs has been of inestimable value in the treatment of chronic sinusitis, one must not neglect to correct surgically, anatomic defects and irreversible pathologic changes in the mucosa which interfere with proper nasal physiologic processes.

PRYOR, Redwood City, Calif. [Am. J. Dis. Child.].

## Pharynx

TUBERCULOSIS OF THE TONSILS IN CHILDHOOD. FRANZ BANHIDY, Schweiz. med. Wchnschr. **79**:376 (April 30) 1949.

Twelve of 200 children 3 to 16 years of age are registered as having primary tuberculosis of the tonsils. All had large tonsils, and tonsillectomy was performed. The presence of tuberculosis of the lungs and abdominal organs was excluded clinically. Frequently, in patients with subfebrile temperatures, swelling of hilar nodes and enlargment of cervical nodes, the possibility of primary tuberculosis of the tonsils must be considered.

AMBERG, Rochester, Minn. [Am. J. Dis. Child.].

## Miscellaneous

ANTIBIOTICS IN THE TREATMENT OF DISEASES OF THE EAR, NOSE AND THROAT. A. C. FURSTENBERG, Ann. Otol., Rhin. & Laryng. **58**:5 (March) 1949.

Furstenberg, after studying the use of antibiotics in otolaryngology, reaches the following conclusions concerning the subject.

Nearly all acute infections of the ear, nose, sinus and throat respond well to the parenteral administration of penicillin. It is not always feasible to isolate and identify each organism, but when prompt clinical response is not noted, the organism must be identified and its sensitivity determined. Penicillin must come in contact with the organism and be sustained there. The author states that penicillin aerosol is of equivocal value in the nose and that the local use of penicillin in the mouth is not dependable except, perhaps, in ulcerative lesions, for which the direct application of cotton tampons saturated with penicillin solution, 10,000 to 15,000 units per cubic centimeter, may be used. The tampons are allowed to remain in contact with the ulcer for twenty minutes or more, four or five times a day. In bronchiectatic cavities, the direct instillation, after aspiration of the secretion, of 2 or 3 cc. of penicillin in saline solution (50,000 units per cubic centimeter) three times a week, is useful. In chronic infections in which there is an encapsulated collection of secretion, or in which thickened mucosa surrounds the area, he feels that penicillin is of little value.

In cases of rhinogenic or otogenic meningitis in which time is an essential factor, steps to identify the organism should be taken and prompt treatment instituted. He advises that both penicillin and streptomycin be given at once, intramuscularly and intrathecally. Even aureomycin and chloramphenicol (chloromycetin®), particularly chloramphenicol, should be used in maximum dosage in conjunction with chemotherapy when an intercellular infection is suspected. Some of these agents can, of course, be eliminated in favor of the specific antibiotic as soon as the causative organism is found and its sensitivity determined. Aureomycin and chloramphenicol are effective when given by mouth; they permeate cell

membranes and seem to overcome the cellular barrier to the spinal fluid. When given by mouth, the concentration in the spinal fluid is equal to 50 per cent of that found in the blood serum.

Furstenberg states that administration of penicillin in the absence of infection may alter the normal basic flora of the throat and that prolonged administration of the antibiotic may produce penicillin-resistant organisms and will adversely affect treatment when it may be sorely needed.

Streptomycin is particularly effective in tuberculosis, cervical adenitis and in tuberculous lesions of the mucous membrane. It is highly probable that its allied agent, dihydrostreptomycin, which is comparatively free from neurotoxic effects, will replace the original antibiotic in the treatment of these conditions.

MILLER, Philadelphia.

STUDIES ON THE TRANSMISSION AND CONTROL OF RESPIRATORY DISEASE WITHIN ARMY BARRACKS: I. HEMOLYTIC STREPTOCOCCAL CONTAMINATION OF THE ENVIRONMENT. C. G. LOOSLI, H. M. LEMON, H. WISE and O. H. ROBERTSON, J. Infect. Dis. **82**:59 (Jan.-Feb.) 1948.

From December 1943 to August 1944, a bacteriologic study was made of dust from floors, beds, wearing apparel and air in the barracks at Camp Carson, Colo. Particular attention was paid to the beta hemolytic streptococcic content. The streptococcic contamination of the barracks was low during the winter and summer and high during the spring.

When cultures from noses and throats contained hemolytic streptococci far greater numbers of the corresponding organisms were found in the bedding than in that of persons with the organisms present only in the throat. In the barracks in which the majority of the cultures from bedding, dust and air containing organisms showed the same type of streptococcus, this same type was most prevalent in the nose and throat cultures of the barrack personnel.

The greatest number of air cultures growing organisms was obtained in barracks in which the beds and floor dust were heavily contaminated with hemolytic streptococci. The highest streptococcic counts per cubic foot of air and the greatest number of cultures growing organisms were obtained during the time of maximum activity, such as getting up, dressing, floor sweeping and bed making. The extensive and uniform contamination of the beds and floor dust can be explained adequately only on the basis of the aerial spread of those organisms throughout the barracks.

TOOMEY, Cleveland [AM. J. DIS. CHILD.].

COMMUNITY SPEECH AND HEARING CLINICS. AMY BISHOP CHAPIN, J. Speech Disorders **12**:331 (Sept.) 1947.

Community speech and hearing clinics reach many children that clinics in public schools do not, and are thus of value in the care of adults as well as of preschool children. The author describes the Cleveland Hearing and Speech Center, affiliated with Western Reserve University and the Cleveland Community Fund.

PALMER, Wichita, Kan.

PRIMARY CARCINOMA OF THE LUNG: CLINICAL ASPECTS. J. N. HAYES, New York State J. Med. **49**:895 (April 15) 1949.

This article was a contribution to the session on diseases of the chest at the annual meeting of the Medical Society of the State of New York, on May 20, 1948. The final diagnosis of bronchogenic carcinoma is usually made by the broncho-

scopist or surgeon or by the pathologist in the mortuary! One must differentiate from hamartoma (a round mass containing calcium surrounded by normal lung tissue) pulmonary tuberculosis, tuberculoma, pyogenic abscess, bronchiectasis, effusion, aneurysm and chronic pneumonia (unresolved). Early signs are few and often absent and fail to give proper warning; later, the symptoms and signs are dependent on the size, location and cell type of the mass. Dyspnea, pectoralgia and wheezing are amongst the principal symptoms. Something may be gained from microscopic examination of smears from coughed-up exudate. "To discover it early the clinician must have the disease constantly in mind."

VOORHEES, New York.

CURE RATE IN LUNG CANCER. RICHARD H. OVERHOLT and IVAN C. SCHMIDT, New York State J. Med. **49**:910 (April 15) 1949.

This report is from the New England Deaconess and the Pratt Diagnostic Hospitals. Cancer is commonly found in the lungs, but until science finds a preventive agent, progress in its treatment must depend on early diagnosis in the preextension stage of the growth (before metastasis). Mass roentgenographic examinations may disclose early cancer before it has gone out of bounds. Then excision, with, of course, removal of all mediastinal glands which can be reached, offers the only hope. With present day technics in the hands of experienced surgeons, the mortality rate is not high and the resultant deformity from excision not too disturbing. A five year survival rate is at hand at this time. The recorded discussions are limited to Dr. Arthur Q. Penta, of Schenectady, and Walter F. Bugden, of Syracuse, N. Y.

Abstracter's Comment: There may have been other open discussions from the floor. It is impossible to abstract the remarks here, but the points made are replete with interest and should be read as published in the *New York State Journal of Medicine*. There were four papers in the symposium, of which this is the final one.

VOORHEES, New York.

DIAGNOSIS AND TREATMENT OF ATRESIA OF THE ESOPHAGUS AND TRACHEO-ESOPHAGEAL FISTULA. ORVAR SWENSEN, Pediatrics **1**:195 (Feb.) 1948.

The increasing number of successful operations to correct atresia of the esophagus and tracheoesophageal fistula has brought renewed interest in this combination of anomalies. The anatomic variations of these malformations can be divided into five groups.

There may be only atresia of the esophagus. The atresia may be associated with a tracheoesophageal fistula to the upper or lower segment of esophagus, or a double fistula may be present. A tracheoesophageal fistula without an associated atresia is rare, but it does occur in about 17 per cent of the cases.

The diagnosis is made on the history of regurgitations of any fluid taken by mouth, followed by bouts of cyanosis. The demonstration of an obstruction in the esophagus by failure of a soft rubber catheter to pass into the stomach is diagnostic.

The babies are prepared for operation by proper hydration and chemotherapy. In practically all patients it is possible to perform end to end anastomosis of the esophagus. After the operation, a gastrostomy is done and feedings are given through this opening until the tenth postoperative day, when oral feedings are begun.

In a series of 32 cases of primary anastomoses of the esophagus there were 5 postoperative deaths.

FROM THE AUTHOR'S SUMMARY [AM. J. DIS. CHILD.].

STENOSIS OF THE ESOPHAGUS DUE TO CAUSTICS. JORGE OTTE, Rev. otorrinolaring. 8:117 (June) 1948.

Otte presents some of the difficulties encountered in esophageal strictures in children caused by the swallowing of caustics. He presents a report on 8 patients who swallowed sodium hydroxide by mistake. Unfortunately, all the patients reported rather late for treatment, and in consequence the benefits of early therapy, in which success in 80 to 90 per cent of cases can be obtained within eight weeks of intensive treatment was automatically altered; in such instances prognosis is usually graver. In the extremely seriously ill patient an early gastrostomy not only improves his nutrition but also permits the insertion of a cord into the esophagus. Retrograde dilation is the preferable method of treatment. However, dilation by mouth can be undertaken in those cases in which the general nutrition is not too badly affected and in which the strictures are not pronounced.

He stresses two important health procedures: (1) the establishment of government control in the sale of caustic products and (2) the establishment of endoscopy centers particularly adapted for children, so that immediate treatment can be instituted with the hope of curtailing the postoperative course for the patient.

PERSKY, Philadelphia.

# News and Comment

## POSTGRADUATE COURSE IN BASIC OTOLARYNGOLOGY

The University of Illinois College of Medicine, department of otolaryngology will give its annual postgraduate course in basic otolaryngology beginning Oct. 2, 1950. A full time program of instruction is followed for those intending to prepare for special practice. The course terminates on June 15, 1951. Further information and application blanks may be secured from the University of Illinois College of Medicine, Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

# Society Transactions

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Oliver E. Von Alyea, M.D., *President*

Lawrence J. Lawson, M.D., *Secretary*

*Monthly Meeting, Dec. 5, 1949*

**Physical Laws of the Mechanism Involved in the Removal of Air From the Respiratory Tract Under Normal and Abnormal Conditions.** Dr. A. C. Hilding (by invitation), Duluth, Minn.

The presenter gave a summary of the currently accepted theory on the mechanism of the production of postoperative atelectasis and pointed out and discussed certain discrepancies in the accepted theory. He described his experiments bearing on the efficiency of ciliary action in handling mucus and on the determination of the cross sectional area of the bronchial tree at different levels. The laws of physics governing the absorption of air from various body spaces were reviewed and illustrated by accounts of the steps involved in the absorption of an air bubble injected into the anterior chamber of the eye at cataract surgery; the absorption of air from a completely blocked sinus; from the middle ear after closure of the eustachian tube by mechanical means, and from an obstructed lobe of a lung. Consideration of the information thus obtained points to something other than absorption operating in postoperative atelectasis to remove the air, and the author stresses the fact that ciliary action is ignored in the production of this condition.

Additional experiments carried out to determine the possible relation between ciliary action and the development of postoperative atelectasis throw some light on the obscurer aspects of this condition. A positive or negative pressure (5 to 40 mm. of water), reaching a maximum in from ten to twenty minutes, was produced in the trachea of a freshly killed hen by a "piston" of mucus moving toward the laryngeal end, motivated by ciliary power. A pressure of 150 mm. of water (comparable to pressures found in atelectasis) was found when several tracheas, with a mucous "piston" in each, were connected tandem; the cumulative effect was manifest in the last trachea. Ciliary mechanism, rather than absorption of air, effected the production of negative pressure in the frontal sinus of an anesthetized, newly killed or newly decapitated dog. Vacuum headache is probably a manifestation of this mechanism. In postoperative atelectasis, when all the air from the affected lobe is removed, the final one of a series of mucous "pistons," pushed up the air passage by ciliary action, becomes stalled, and atelectasis is complete. Absorption also plays a role, depending on such things as comparative partial pressures in the venous blood and in the alveolar air, solubility rates, diffusion rates and the presence of interposed membranes.

The conclusion is reached that absorption of air from the various body structures described occurs according to the known laws of physics. Air apparently can be completely absorbed from almost any space or tissue in the body, provided that sufficient time is allowed and that the space containing the air can collapse. If the air-containing space cannot collapse, or can do so only partially, a negative pressure develops, depending on the gas pressures present in the absorbing fluid.

In obstructed portions of the lungs, in the sinuses and probably in the middle ear, another mechanism is involved in the removal of air, namely, ciliary action and moving masses of mucus. The negative pressure in postoperative atelectasis is probably maintained solely by ciliary action.

DISCUSSION

DR. PAUL HOLINGER: First, I should like to congratulate Dr. Hilding on this fundamental research work that continues the excellent studies he has already published on this and allied subjects. Anyone who has performed a bronchoscopy on a patient with postoperative massive collapse of the lung, realizes that the textbook picture of a mucous plug obstructing a major bronchus should be discarded. Mucus is produced in quantity throughout the tracheobronchial tree, and there is almost as much mucus on the so-called uninvolved side of the chest as on the side that is collapsed. No single plug is ever demonstrated as such. I wonder if Dr. Hilding's experiments can explain the rapidity of development of postoperative atelectasis in some cases—for example, those that occur on the operating table during the surgical procedure. We have roentgenograms that show that rapid development, and just as rapid clearing, follow adequate aspiration. I believe the secretions themselves can replace a great deal of the air in the bronchi and lungs, just as occurred in some of Dr. Hilding's experiments on the sinuses of dogs. Possibly this mechanism and that described by Dr. Hilding work in combination to bring about the final clinical picture.

DR. THOMAS C. GALLOWAY: We members are very fortunate to hear this presentation by Dr. Hilding, not only for the actual scientific data he has given us but as an example to young men who, with all their fine laboratory facilities, complain that they are not able to do good experimental work. I think it would interest them to know that Dr. Hilding does this work practically on the kitchen table, with his son assisting him, procuring his own material with practically no facilities except that which he improvises.

This is a difficult paper to discuss without having had a chance to sit down with a slide rule to figure out some of his computations and to analyze them more completely. His ideas are scientific. There are two ways of approaching the subject: There are some interesting essays Dr. Hilding would like to read, and there are formulas developed from models. There is one formula that may explain some obstruction in the divided lumens of the respiratory tract. It is not the same thing to say that the lumen of the upper trachea which has volume will have the same resistance as the lumen which is much more diffuse. Roughly, the response to the flow of material can vary from solid plasma to gas as light as helium. The response is something like the fourth power of the diameter, increasing, of course, inversely as the lumen narrows. I think one must consider the secretion in the bronchioles where the plug is developed as resulting in atelectasis. That is important. It is quite a different thing whether those plugs are in the bronchioles or whether they are secretion. I think that, as in poliomyelitis, where the obstruction is not a mucous plug but a rather thin secretion the picture must be quite different. I like to go back to the experiment of Steinberg, in which rubber plugs were put in the bronchi and bronchioles, demonstrating that atelectasis developed within twenty-four hours and that it became irreversible in forty-eight to seventy-two hours. It is fine to get this down to a simple formula but when there is obstruction, there are circulatory changes, which add to the picture of obstruction, congestion and edema. This is something I hope Dr. Hilding does analyze.

This is certainly a great advance in a subject about which very little that is really scientific is known. Incidentally, I think we all have seen plugs which we have taken out with the bronchus. I remember that in one case there was probably 3 cc. of very tenacious material in the left bronchus, removal of which relieved the atelectasis immediately. I think we have seen these plugs in tracheobronchitis. We determine that there is no air going through; we irrigate and get out the plug, and immediately we find that air is going in. I feel that although we do not always see the plugs they are likely to be important, but they are likely to be smaller conglomerations of a thinner secretion. We should be grateful to Dr. Hilding for this excellent presentation.

DR. JOHN BALLENGER: I, too, enjoyed Dr. Hilding's paper and should like to make two comments. He made the statement that it takes five or six days for air injected into the anterior chamber of the eye to be absorbed, and perhaps even longer for air trapped in the sinuses or middle ear to be absorbed. Van Dishoeck's work with the pneumophone suggests that a shorter period is required for air trapped in the middle ear to disappear. In a person with a persistently negative pneumophone reading he restored the middle ear pressure to normal by catheterization of the eustachian tube and then noted that it was only a matter of hours until the original negative pressure was regained.

The other point that occurred to me was with regard to asthmatic persons. In the postmortem examinations in some cases, atelectasis is usually not found, even though the alveoli and terminal ramifications of the bronchial tree are completely obstructed with mucus. I am familiar with the fact that some of the cilia have been destroyed by the disease process, but there are other cases in which the ciliary apparatus is intact; there is a great deal of obstructing mucus, and still atelectasis is not found. Perhaps this is explained by the anatomic phenomenon of alveolar pores. These are vents or connections between adjacent alveoli which permit exchange of air and other gases between the alveoli. It has been shown by a number of people who have worked on development of postoperative atelectasis that obstruction must occur at such a level that an entire lobe is obstructed. If the obstruction is at a lower level the air can sidetrack the obstruction by way of the alveolar pores, and atelectasis does not occur.

DR. ANDERSON C. HILDING: I wish to thank the discussers for their kind and illuminating remarks. Dr. Holinger pointed out that air could very well be displaced by the thin secretion which is often produced in anesthesia. This, I believe, is not only a possibility but a probability.

What Dr. Galloway said about increase in friction with decreasing diameter is indeed true. It is apparently on this account that the arterial tree increases its total stream bed very rapidly as the diameter of the vessels grows smaller. If the capillaries were not 800 times the size of the aorta the heart could not force the blood through. In the bronchial tree the situation is somewhat different. The friction factor is much less; the rate of flow of air is comparatively low, and the flow is to and fro. For these reasons, the total cross sectional area does not need to be so great as in the arterial tree. As he pointed out, it is a fact that the thin secretions produced in poliomyelitis are removed very slowly by ciliary action. This is also true in influenza.

**Fifty Years of Scientific Progress in Otolaryngology.** DR. ALFRED LEWY.

**Outstanding Personalities of the First Half Century of the Chicago Laryngological and Otological Society.** DR. SAMUEL SALINGER.

**Oliver E. Van Alyea, M.D., *President***
**Lawrence J. Lawson, M.D., *Secretary***
***Monthly Meeting, Jan. 9, 1950***

**Inner Ear Deafness of Sudden Onset.** Dr. Jacob J. Zuidema (by invitation) and John R. Lindsay.

Sixteen cases have been presented of unilateral inner ear deafness of sudden onset with or without vestibular symptoms. The first 4 cases represent types in which the aural complication was associated with a systemic disease.

The next 12 cases of unilateral, sudden deafness in the inner ear with varying degrees of vestibular disturbance are of uncertain cause. It cannot be assumed definitely that in all 12 cases the cause was the same, but the similarity of the clinical picture strongly suggests that the deafness was the same type.

Vascular diseases such as hemorrhage, thrombosis and occlusion, vasomotor disturbance, allergy and toxic neuritis were all considered as being the possible cause. Toxic neuritis must be seriously entertained as the etiologic factor, because of the high incidence in young adults less than 30 years of age in the absence of chronic systemic disease.

The last 3 cases demonstrate a specific syndrome of bilateral inner ear dysfunction, associated with an inflammatory ocular disturbance.

DISCUSSION

Dr. Francis Lederer: If one were to take a census of this audience, I dare say everyone in practice could find among his patients 16 cases which would typify the representative ones reported. The only thing we would have to make sure of was that we were not all reporting the same cases, because patients of this type are the ones who go from one otologist to another seeking reassurance: "Will this happen to my good ear?" And you, stroking your chin and looking out of the window, say, "No, in my experience it will not happen in the other ear." You are not much of a prophet, because later these patients come in with tinnitus and, under your very eyes, as it were, total deafness develops. You use some terms like "virus" disease, "allergy," "hemorrhage into the labyrinth" or mention etiologic factors for which you have not much proof. In such cases I always make roentgenograms of the internal meatus. I am looking for an acoustic neuroma, but by and large these are not patients on whom one can make a specific diagnosis.

I wish we had some temporal bones to study in this series. In this disease, whatever its cause, one deals with an irreversible reaction, and I feel like a minister making a divine wish that the same thing will not happen to the other ear. I had hoped that Dr. Lindsay and his associates would bring us something of value in the management of this condition. I give thiamine hydrochloride, neostigmine, histamine, antihistamine for the psychogenic influence on the patient, but their value has been therapeutically insignificant.

Dr. Thomas C. Galloway: I wonder if the last cases cited might fall into the classification of Cogan's syndrome of interstitial keratitis with auditory and vestibular disturbance. I recently saw a young woman with loss of hearing in both ears and recurring keratitis said to be due to this unusual disease. I am assured that the prognosis is grave.

**Deafness Following Trauma to the Head: A Clinical and Experimental Study.** DR. HAROLD F. SCHUKNECHT (by invitation).

Diminished hearing acuity may follow a blow to the head. In some of these cases there is a fracture of the temporal bone. The fracture may be either longitudinal or transverse to the long axis of the petrous pyramid.

A longitudinal fracture of the temporal bone is characterized by bleeding from the ear, rupture of the tympanic membrane and a combination of conduction and nerve deafness. The conduction deafness is caused by direct injury of the middle ear structures and is partially or completely reversible.

Following a transverse fracture there is severe vertigo with nystagmus to the opposite side, hematotympanum, complete deafness and total loss of vestibular function in that ear. The fracture line extends through the inner ear, and degeneration of the membranous labyrinth occurs.

Deafness may also occur in an ear in which there is no clinical or roentgenologic evidence of fracture of the temporal bone. The hearing loss is of the nerve type and is severest for the high tones, particularly the 4096 frequency. In severe injury all frequencies may be involved. This type of deafness is frequently seen in the ear on the side opposite a fracture of the temporal bone.

Brunner, Stenger, and Wittmaack have shown in animal experiments that hemorrhage often occurs into the perilymphatic spaces. Bleeding into the auditory nerve is less common. Wittmaack found degeneration of the hair cells and spiral ganglion to be severest in the middle cochlear coil in cats subjected to blows on the head. He conjectured that a traveling pressure wave injured these structures directly.

A number of human temporal bones have been described in the literature of cases of deafness following head injury without fracture. In some cases there have been shown connective tissue and bone in the perilymphatic spaces with degeneration of Corti's organ and the spiral ganglion. In several have been shown only severe degeneration of Corti's organ and the spiral ganglion. Isolated chip fractures may also occur.

The case histories and audiograms in 7 cases were presented.

My interest was mainly in determining the nature of the pathologic condition accounting for the nerve deafness in such cases, so-called inner ear concussion.

The problem was studied in cats conditioned to pure tone stimuli so that hearing tests could be performed on them. These animals were subjected to head blows while under anesthesia. All degrees of deafness were produced. The 3000 to 8000 frequency range was most severely involved, paralleling that found in the human being.

The histologic studies of these animals are not complete and will be reported in detail at another time.

DISCUSSIONS

DR. SHERMAN SHAPIRO: There have been quite a number of experiments, beginning with those of Wittmaack, to show the histologic changes following a blow on the head in guinea pigs and other animals. These are the first I know of in which conditioned reflexes were used. I notice that Dr. Schuknecht mentioned what happens in the cat, and I should like to caution against translating this too literally in terms of human beings. I have had a large experience in handling head injuries, all studied from the acoustic and vestibular standpoint. My routine is to make tuning fork tests with audiograms on certain patients, and my experience is that not more than 5 or 10 per cent show longitudinal or cross fractures or anything involving the peripheral endings of the eighth nerve. Less than 5 per cent complain of hearing difficulties, but nearly all, in the first few months, complain of dizziness. I have found a number of times pure concussion deafness without

evidence of fracture, but that is quite exceptional. The objective findings as a rule are postural nystagmus and some imbalance of the central vestibular apparatus.

I wonder about the author's case of deafness following the use of the mallet and chisel on the mastoid process. In the old days when the mallet and chisel was used very largely for mastoid operations, I never had patients complain of subsequent deafness. I never found the trauma sufficient to injure the inner ear. Two of the patients discussed had symmetric curves, quite suggestive of otosclerosis, and I think they should not be accepted as traumatic. One must remember the difference between the skull of a human being and that of a laboratory animal. Certainly, few of my patients with concussion without fracture led me to believe, from their complaints or from the cochlear findings, that they would show any such pathologic change as indicated by a blow on the head in these animals.

Dr. Paul A. Campbell: I have nothing to add from the viewpoint of either military or aviation aural trauma. One of Dr. Schulknecht's cases interested me, however, from another point of view. It was the case in which the spondee word response level was about 20 decibels above the pure tone threshold level. I believe this type of discrepancy in a test should lead one to consider possible psychogenic origin. Of course, it is well known that head injuries often lead to psychogenic disorders. If discrimination tests are performed on such persons, the hearing is often found to be much poorer than one would suspect from pure tone thresholds.

Dr. Alfred Lewy: I do not know whether or not I understood Dr. Shapiro correctly in saying that he did not always use the audiometer in cases of trauma of the head. He said patients usually complain of vertigo rather than of loss of hearing. When they do complain of loss of hearing and it is not complete, it is difficult to determine whether or not it is indicative of trauma, except when they show greater loss at about 4096 cycles. That has been pretty well established as quite characteristic of trauma to the internal ear. Whether or not the recruitment phenomenon is going to be a definite point in diagnosing deafness, I do not know. I recently read a report on a series of cases of sudden deafness, all of which were attributed to the inner ear; in them the recruitment phenomenon failed to be seen. Perhaps the recruitment phenomenon may apply only to certain types of inner ear deafness.

I agree with Dr. Shapiro that the mallet and gouge used in mastoid surgery did not result in permanent damage to the ear. Patients sometimes complained of headache, but no one used a one pound hammer or a dull instrument, so one can hardly compare data on them with the experimental data reported.

As to prognosis in vertigo following head injury, it is generally good in young people. If it has persisted more than six months, and in patients beyond middle age, it is not good.

Dr. Harold F. Schuknecht: Several of the clinical cases were unusual and interesting and your comments on these cases are interesting. Case 16 was that of the young boy who had a severe dip in the 4096 frequency following a mastoidectomy. There was fairly good high tone hearing prior to operation. A dense, sclerotic mastoid cortex was removed with a mallet and gouge. I do not imply that this type of injury is common, but believe it to be rare. That the deafness was the result of a shock-pulse injury seems very likely. The other patients under discussion, mostly young persons, all heard well before the injury and noted deafness afterward. Careful audiograms in a soundproof room are essential to the proper evaluation of hearing in all persons with head injury. Case 14 was that of a 54 year old woman with a rather flat hearing loss of between 30 and 50 decibels in both ears following a head injury. A striking discrepancy was the relatively good score on the spondee word test. I agree with Dr. Campbell that

the possibility of psychogenic deafness exists in this one case. Repeated audiograms were remarkably consistent in this case. That any of these patients might have otosclerosis, Ménière's disease, etc., seems beyond the realm of possibility.

**Some Unusual Mixed Tumors of the Nose and Throat.** DR. HENRY B. PERLMAN.

The so-called mixed tumors of the salivary and mucous glands are of special interest to the otolaryngologist. Although the term is widely used it often represents an obscure clinical and pathologic entity, especially when applied to tumors found away from the major salivary glands. Because of the relatively high incidence in the parotid gland, this diagnosis is readily considered when dealing with a tumor here, and the clinical and pathologic picture is more generally agreed on. They are much less frequent in the sublingual gland, occurring about 1 to every 100 cases involving the parotid gland. These tumors do occur in such places as the mouth, tongue, soft palate, nose, sinuses and pharynx. In these less common locations, a diagnosis may be suspected when one is confronted with a slowly expanding, rubbery, lobulated, nonpainful, nonmetastasizing encapsulated tumor. New and Withridge collected more than 60 tumors in the soft palate and pharynx alone, while Owens found reports of several hundred during the last twenty years, with the palate and nose being the commonest site. There is a problem of preliminary biopsy too. One may hesitate to open the capsule for fear of spreading tumor cells. Furthermore, a small specimen may not reveal the true nature of the lesion, which may have benign and malignant characteristics histologically in different parts. These tumors may show bone destruction on roentgenologic examination that resembles that seen in infiltrative carcinomas, but may be found at operation to be due to slow resorption of bone on account of pressure, as well as by definite infiltration. The tumor may remain unchanged in basic character while growing to enormous size. This is commonly seen in parotid tumors. The facial nerve may be slowly stretched over such a tumor, as over an acoustic neuroma, without disrupting its function. Since they are as a rule radioresistant, the correct diagnosis guards against unnecessary radiation, too radical surgery or a very grave prognosis. The surgical problems concerning the identification and preservation of the facial nerve when dealing with the parotid tumor were well described recently by Buxton, Maxwell and Cooper. The use of a nerve stimulator to help identify some of the facial nerve branches at operation is also worth emphasizing.

The surgical attack is less radical than that needed for an infiltrative nonencapsulated tumor, but the capsule may be very thin in some places or, indeed, missing. The capsule should be very carefully handled to prevent opening into the tumor, which grows from its inner surface. The capsule must be cleanly removed with the tumor to affect a cure.

Clinically, this is a peculiar tumor in that it may show long periods of quiescence, remaining a given size for many years and then growing more rapidly. The longest period in my group was about thirty years. Similarly, after removal, years may elapse—ten to twenty years is not unusual in my records—before recurrence is noted. The recurrence is not necessarily of a more malignant nature either clinically or pathologically and may be localized without regional gland involvement. Several recurrences may take place in this manner. On the other hand, recurrences may take place in ever more frequent sequence after many years of apparent cure, leading finally to local and to distant metastases, with fatal termination. This is a peculiar tumor histologically, the character of which has long been disputed, but which is now considered by most competent pathologists as

arising only from epithelial cells in the gland. A single case may show many different kinds of architecture, but, on the other hand, may be quite homogenous in its cell structure. The term "mixed cell tumor" or "mixed tumor" is really inappropriate and stems from the belief that the cartilage-like stroma in some of these cases represented a connective tissue participation as well as an epithelial cell origin. These cartilage-like areas in the stroma are the result of mucous formation, probably by the tumor cell. The tumor cells may be widely separated in some areas of this mucoid stroma and give the appearance of immature cartilage. Another term that has been used to describe the histologic picture of some of these glandular tumors is "cylindroma," a term that is also of little significance for a basic understanding of these cases and used by different authors with different implications. The term may be used to describe the solid cords of cells seen in some of these tumors that resemble cylinders, while another meaning referred to the presence of a thick collar of pink-staining collagen around the columns of tumor cells.

Four cases are described in detail, 1 arising in the soft palate, 1 in the sublingual gland and 2 in the antrum and floor of the nose, probably arising from the inferior turbinate body.

Certain modifications of the lateral rhinotomy operation used in the last 2 cases are described.

DISCUSSION

Dr. Stanton A. Friedberg: We are deeply indebted to Dr. Perlman for this presentation. It is a live and important subject to otolaryngologists because we are, by and large, the first to see these patients. This is the type of tumor that evokes more difference of opinion among pathologists than almost any other lesion in the human body.

As Dr. Perlman mentioned, there is apt to be great variation in the histology in different parts of the same tumor. The combination of epithelial and connective tissue elements has led to their being designated as mixed tumors, but we often see such references to them as adenoid cystic carcinoma, cylindroma, basal cell carcinoma, myxochondrocarcinoma and mucoepidermoid tumor of salivary gland origin. Although some of these lesions may be encapsulated, this is not universally true and capsular boundaries are apt to disappear as the growths increase in size. Whereas some may be judged as being histologically benign, the clinical behavior certainly demands that we consider them as potentially malignant, in view of their marked tendency toward local recurrence and their reactivation after periods of quiescence. For this reason it seems to me that the classic intranasal and sublabial antrotomies are completely out of order in the surgical approach to these lesions when they involve the maxillary sinus.

Here is a lantern slide showing the operative defect in a girl who had had multiple limited operations for a mixed tumor of the palate over a period of eleven years. One year ago a resection of the maxilla was performed, and, whereas my associates and I are dubious as to the eventual outcome, we feel that for the first time a truly adequate removal was effected. A prosthetic device allows her to eat and speak normally but permits of ready access to the cavity at all times. In my opinion, the initial attack on these tumors will determine the outcome, and I should like to make a plea for their radical removal at the first operation.

Dr. Maurice F. Snitman: The term "mixed tumor" is inappropriate, inasmuch as it does not give a true histologic picture of the lesion and does not sufficiently stress malignancy.

Histologically, the tumor is an epithelial structure, the presence of cartilage-like or myxomatous tissues within it being the result of a mucoid transformation of the stroma by the secretion of the acini of the tumor. The inclusion of this tumor in the adenocarcinoma group could be considered proper, in view of its well known malignant character.

When the tumor is located in the nasal cavity or antroethmoid area, I use the sublabial approach rather than the external facial incisions. It is imperative that a good portion of the hard palate of that side be removed so that a patent opening is obtained through which the cavity may be carefully observed postoperatively. These tumors are malignant and will recur if incompletely removed.

Dr. Thomas C. Galloway: We physicians are likely to try to include in one category too many types of tumor. They cannot always be classified too closely. The tumors which arise in the sinuses, called by Ringertz salivary cell tumors, behave differently from those oftener seen in salivary glands or soft tissues. The tumor is rather uniform grossly, and histologically it is likely to be encapsulated and destroys bone by erosion rather than invasion; it does not greatly change its histologic structure, except as modified by such factors as pressure and infection.

The interesting thing about the mixed tumor is that when cancerous changes develop it appears to come from multiple foci. It may remain relatively latent for years and then rather rapidly begin to grow from these numerous centers. Has the speaker any explanation?

I hope no one considers the advice to do a radical operation a direction to do slashing surgery on gland tumors. The dissection must be performed most painstakingly. The capsule is very thin, and if it is torn recurrence is likely, with subsequent failure; yet, for removal, dissection must be carried to this capsule.

Ten years ago I removed a so-called myxoma from the parotid gland of a 20 year old woman, with no recurrence to date. Her identical twin died a few years earlier after operation on a parotid tumor. Could these tumors have arisen from one anlage?

Dr. Samuel Salinger: I should like to add one or two points. In my experience the tumor in which the capsule is ill defined and in which the epithelial cellular elements predominate is the one in which malignant degeneration or recurrence is likely. The five year limit is not always the outside limit. Quite recently one of my patients had a recurrence in the same location after eight years. As a rule, tumors in the upper jaw usually recur locally; whereas tumors of the mandible are more likely to metastasize.

The point Dr. Galloway brought out is, I think, very appropriate. There is a variety of tumors bearing various names according to the predominating elements, and yet genetically, all are the same. The important thing is not the name but the recognition of the potentiality of the growth.

Dr. Henry B. Perlman: The question of the amount of resection one must practice in these tumors is difficult to settle, but I think one would approach a tumor like this less radically than one which is wild from the onset, and, while I had to be radical to the extent that the exposure technic in the last two cases was radical, the actual tissue removed was limited to the tumor itself, rather than to a margin of normal-looking tissue, as in infiltrating carcinomas.

The terminology of these tumors is very confusing, and I would not attempt to commit myself on the histologic sections. We physicians depend on the pathologist, who has cooperated with us for many years; we feel his judgment has great merit and have come to depend on it because he knows the clinical course of these cases almost as well as we do. We depend on him to point out histologic features which may be clinically significant.

Leaving an opening into the mouth might be the soundest policy, certainly in attacking one of the infiltrating nonencapsulated antral carcinomas in which recurrence comes from multiple areas. The reason for preserving the integrity of the mouth cavity in this third case was that the tumor appeared to be a circumscribed encapsulated one. So far as the histogenesis is concerned, the more one reads about these tumors the more one realizes that it is a specialized field of histopathologic interpretation; a clinician may rightfully hesitate to make a statement about the histologic sections. A clinician may have ideas about the histology, but a trained pathologist with a large experience with the clinical and pathologic nature of tumors in this field can render valuable aid. After obtaining consultation with the pathologist and the radiologist, the clinician must make the ultimate decision as to the best method of procedure in any given tumor.

It is not known why these tumors may remain latent for ten or twenty years and then start growing. The recurrence may not be more malignant in character than the original tumor.

Neural infiltration is one of the histologic features that suggest a method of spread of tumor that is dangerous to the patient. Obviously, five year cures do not mean anything in mixed tumors.

# Book Reviews

**Bronchography.** By Eelco Huizinga, M.D., and G. J. Smelt, M.D., Department of Otorhrinolaryngology, University of Groningen. Cloth. Price, 39 florins. Pp. 270, with 146 illustrations. Van Gorcum & Co., N. V., Aan Den Brink, Assen, Netherlands, 1949.

This work presents the subject which is gaining great impetus because of the necessity of an accurate diagnosis for the proper treatment of pulmonary diseases. The subject matter is systematically presented as follows: Chapter 1 presents the anatomy of the bronchial tree and the lung segments. Chapter 2 is devoted to the physiology of the bronchial tree, in which only the main points of importance with regard to bronchography are discussed. Chapter 3 contains general considerations on bronchography and a survey of the literature concerning its development. Chapter 4 presents the roentgenologic anatomy of the normal bronchogram. Chapter 5 is concerned with the interpretation of the bronchogram and presents a description of the way in which serial bronchograms should be read. Chapter 6 is devoted to the pathologic bronchogram, and chapter 7 contains the technic of bronchography as practiced in the Groningen University clinic.

The paper, printing and illustrations are good. A few minor points may be mentioned in passing: On page 68, "radical" traction should be replaced by "radial" traction. The authors' routine of performing bronchography before bronchoscopy is contrary to that of most clinics in this country. Also, the recommended use of tetracaine hydrochloride (pontocaine hydrochloride®) rather than cocaine is contrary to recent studies.

This book is recommended reading for all physicians interested in the problems referable to the bronchopulmonary tract and their diagnosis.

**Les suppurations chroniques de l'oreille moyenne.** By Robert Maduro, Otorhinolaryngologist, Hospital of Paris. With the collaboration of C. Chausse, Alain Gaston and Henri Henrot. Paper. Pp. 344, with 33 illustrations. L'Expansion scientifique francaise, 23 rue du Cherche Midi, Paris 6, 1949.

This book, one of recent monographs and imported from Europe, demonstrates in a fine way the caliber of work and intellectual activity that is taking place in the medical centers of France. The book is systematically arranged by the author and his collaborators as follows: introduction; anatomy; bacteriology; pathology; cholesteatoma; eustachian catarrh; suppuration of the attic, antrum and tympanum; complications; diagnosis; roentgenologic studies, and treatment, general, medical and surgical. Thus, chronic suppuration of the ear is adequately covered. The illustrations supplement well the material in the text.

**Névralgies du Glosso-Pharyngien.** By Maurice Deparis, Associate Professor of the Faculty of Medicine of the University of Paris, Physician, Hospital of Paris. Paper. Price, 360 francs. Pp. 154, with 5 illustrations. Masson et Cie, 120 Boulevard Saint-Germain, Paris 6, 1949.

This book on neuralgias of the glossopharyngeal nerve is a successful attempt to present to practitioners a clear and concise picture of the subject matter. The contents are arranged in several large sections with well planned subdivisions,

so that the material is logically and adequately presented. Anatomy and physiology are covered first, then are described different clinical pictures and the diagnostic problems involved, based on the author's personal experience. The different medical and surgical therapeutic measures are then presented. The book has an extensive bibliography. For one able to read French and interested in otolaryngology, this book is recommended for the viewpoint it presents.

**Life-Situation-Speech-Reading as a Clinical Treatment.** By Lucelia M. Moore, M.S.

This booklet is from a paper which was presented at the American Speech and Hearing Association in December 1949. It is an excellent explanation of the new concept of speech reading. The advantages of teaching speech reading over the former methods of teaching lip reading are very comprehensively and lucidly demonstrated. The method has been adopted by most speech and hearing centers, especially by those in the Armed Forces. The philosophy of speech reading and treatment of the hard of hearing patient as a whole is brilliantly explained. Moving picture films, which have been prepared for the specific purpose of instruction in speech reading, are carefully described, and illustrations from excerpts are presented. The discussion here is extremely worth while and well presented.

Copies of this booklet may be obtained from Lucelia M. Moore, Instructor of Speech Reading, Speech and Hearing Clinic, University of Southern California.

## NOTICE

The index for Volume 51 will be mailed with the July 1950 number.

# Directory of Otolaryngologic Societies *

## NATIONAL

### American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. William H. Johnston, 1515 State St., Santa Barbara, Calif.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit 26, Mich.
Place: San Francisco. Time: June 28-30.

### American Academy of Ophthalmology and Otolaryngology

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles 14.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 8-13, 1950.

### American Broncho-Esophagological Association

President: Dr. Leroy A. Schall, 243 Charles St., Boston.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-26, 1950.

### American Laryngological Association

President: Dr. Ralph A. Fenton, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Hotel Mark Hopkins, San Francisco. Time: May 23-24, 1950.

### American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 25-27, 1950.

*SECTIONS:*

Eastern.—Chairman: Dr. Robert L. Moorhead, 125 Remsen St., Brooklyn, N. Y.
Southern.—Chairman: Dr. William D. Stimson, 899 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. J. Marion Sutherland, 3001 W. Grand Blvd., Detroit.
Western.—Chairman: Dr. J. B. Naftzger, 6777 Hollywood Blvd., Hollywood, Calif.

### American Otological Society

President: Dr. Philip E. Meltzer, 20 Charlesgate, W., Boston.
Secretary: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 3, N. Y.
Place: Hotel Mark Hopkins, San Francisco. Time: May 21-22, 1950.

### American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Samuel F. Kelley, 47 E. 61st St., New York 21.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.

### American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. George E. Shambaugh Jr., 55 E. Washington St., Chicago.
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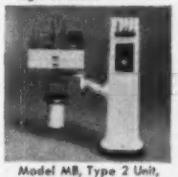

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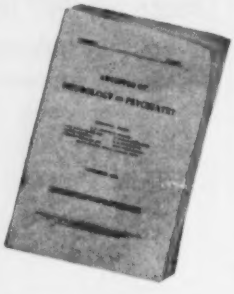

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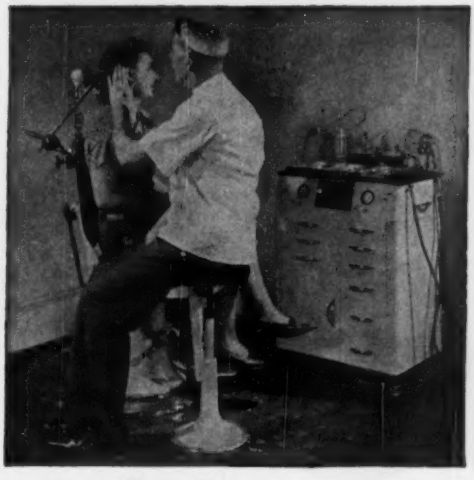

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# CONTENTS


PAGE

**Histamine in Treatment of Certain Types of Headache and Vertigo Following the Fenestration Operation.** George E. Shambaugh Jr., M.D., Chicago ..... 781

**Use of Speech Tests for Evaluation of Clinical Procedures.** S. Richard Silverman, Ph.D., St. Louis ..... 786

**Clinical Application of Bone Conduction Audiometry.** Raymond Carhart, Ph.D., Evanston, Ill. ..... 798

**Masking and Shadow Hearing in Bone Conduction.** Maurice Saltzman, M.D., and Matthew S. Ersner, M.D., Philadelphia ..... 809

**Hoarseness in Children.** John M. Lore Jr., M.D., New York ..... 814

**Streptomycin in Treatment of Tuberculous Otitis Media: An Evaluation.** Leon L. Titchie, M.D., Tucson, Ariz. ..... 826

**Diagnosis of Intracranial Lesions by Vestibular Tests.** Fernando G. Santamarina, M.D., Habana, Cuba ..... 831

**Histologic Picture of Inherited Nerve Deafness in Man and Animals.** Franz Altmann, M.D., New York ..... 852

**Transantral Approach for Excision of Sphenopalatine Ganglion for Intractable Facial Pain.** David L. Poe, M.D., New York ..... 891

**Otosclerosis: Résumé of the Literature with Clinical Observations.** V. S. Subramanian, M.D., M.Sc. (Med.), F. A. C. S., Madras, India .. 901

**Thrombosis of the Cavernous Sinus.** Will P. Pierce, M.D., Baton Rouge, La. ..... 917

**CASE REPORTS:**

**Tumor of the Glomus Jugularis.** D. A. De Lisa, M.D., Schenectady, N. Y. ..... 925

**CLINICAL NOTES; NEW INSTRUMENTS AND TECHNICS:**

**Ear Aspirator and Injector.** Barnard C. Trowbridge, M.D., D.Sc. (Med.), Kansas City, Mo. ..... 928

**ABSTRACTS FROM CURRENT LITERATURE** ..... 930

**NEWS AND COMMENT** ..... 935

**SOCIETY TRANSACTIONS:**

**Chicago Laryngological and Otological Society** ..... 936

**BOOK REVIEWS** ..... 946

**DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES** ..... 948